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"In writing 'A System of Clinical Medicine' Dr. Savill has adopted a plan which we believe will be found of great assistance both to students and to practitioners. He has approached the subject from the standpoint of symptomatology. . . . This scheme has been admirably carried out and for the most part Dr. Savill has been successful in producing a very useful and practical work . . . at the end of the volume are a large number of useful formulæ for prescriptions. . . . We have formed a high opinion of Dr. Savill's work; we wish it the success that it deserves." —*The Lancet.*

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WORKS BY THE SAME AUTHOR.

TRANSLATION OF PROFESSOR J. M. CHARCOT'S CLINICAL LECTURES ON DISEASES OF THE NERVOUS SYSTEM. New Sydenham Society, 1889.

ON A NEW FORM OF EPIDEMIC SKIN DISEASE. London, 1893.

CLINICAL LECTURES ON NEURASTHENIA. Second Edition. London, 1902.

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A SYSTEM
OF
CLINICAL MEDICINE

DEALING WITH THE

DIAGNOSIS, PROGNOSIS, AND TREATMENT OF DISEASE

FOR

STUDENTS AND PRACTITIONERS

BY

THOMAS D. SAVILL, M.D.LOND.

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VOLUME II

*CERTAIN GENERAL DISORDERS; DISEASES OF
THE SKIN AND THE NERVOUS SYSTEM*

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PREFACE TO VOLUME II.

THE design and objects of this work are set forth in the Introduction to the first volume, which dealt with the symptoms of Local Diseases and Pyrexial Disorders. This, the second and completing volume of the book, comprises chapters on General Debility, Pallor and Emaciation; symptoms referable to the Extremities; symptoms referable to the Skin; symptoms referable to the Nervous System; and, finally, an epitome of Clinical Bacteriology and the Examination of Pathological Fluids. This volume, like the first, is separately indexed.

I am indebted to Dr. Purves Stewart for criticism of the proofs on the chapter on the nervous system, and similarly to Dr. R. H. Cole in connection with mental diseases, to Dr. F. J. Poynton in connection with rheumatic fever, to Dr. Lloyd Tuckey in connection with hypnotism, to Mr. Evan Evans for general assistance in proof-reading, and to Dr. Vickerman Rutherford and my wife, Dr. Agnes Savill, for the abbreviation and revision of the chapter on diseases of the skin. To the last named I am also indebted, as previously mentioned, in many other ways.

Any criticisms or suggestions which would help to make the work more useful to those for whom it is primarily intended—the practitioner and senior student—would be gladly welcomed.

THOMAS D. SAVILL.

60, UPPER BERKELEY STREET, LONDON,
March, 1905.

*

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A SYSTEM
OF CLINICAL MEDICINE.

CHAPTER XVI.

GENERAL DEBILITY, PALLOR, EMACIATION.

A FEELING of general weakness and lassitude is a symptom common to a great many diseases, but we are now concerned with those in which this is the only obvious, or at least the most prominent, symptom for which the patient seeks relief. Diseases in which debility is the chief symptom may be classified clinically into two great groups according to whether they come on acutely and are attended by pyrexia, or not. Debility coming on acutely with pyrexia was fully dealt with in the preceding chapter. There still remains a large group of diseases which we shall now have to consider in which the weakness is of gradual onset, runs a chronic and indefinite course and is unattended for the most part by any notable elevation of temperature; and these diseases may be attended by anaemia, or by emaciation. Here we shall often meet with the beginnings of disease, beginnings which may however lead to a serious and fatal issue. It is therefore of the highest importance that an exact diagnosis should be made, and treatment adopted as early as possible.

The debilitating conditions mentioned in this chapter may be unattended by any other symptom, or only by the pallor of anaemia or the wasting of malnutrition, and many give rise to no characteristic anatomical changes after death. Their pathology in some instances is extremely obscure, and its elucidation in the future must largely depend upon the co-operation of the analytical chemist with the physiologist and the physician, a large proportion of them being undoubtedly due either to some kind of autotoxic, hæmolytic, hæmogenitic, or other blood changes on the one hand, or to malnutrition and a profound disturbance of metabolism on the other.

PART A. SYMPTOMATOLOGY.

§ 394. General Debility. Malaise, lassitude, inability to complete a day's work, are some of the terms used to describe the symptom under consideration, which is essentially chronic in its course. The weakness is generalised, and it may affect the mind as well as the body, for there is not only a disinclination to take muscular exercise, but an inability to concentrate the attention or accomplish mental work. The weakness may vary in kind and degree from very slight malaise to a total incapacity to move. Many diseases in this category are apt to be overlooked in their earlier and more curable phases. The patient may attribute his ailment to "slight digestive derangement," or think he has "been working too hard" or "wants a change," and perhaps he calls on his doctor "as he was passing" just to confirm his own diagnosis and "give him a tonic." These cases may tax the young practitioner's skill and tact in several ways. Fresh from studying instances of marked diseases in hospital, he may regard these cases as trivial and "uninteresting"; and even if he detects the beginning of some insidious malady the patient may meet his suggestion of serious ailment not only with surprise, but even with resentment and distrust. Some tact therefore is required, and the practitioner may find it wise to place himself in communication with some discreet friend or relative of the patient.

Fallacies. The distinction of general debility from *paralysis* is not usually difficult, though patients with multiple peripheral neuritis, early paraplegia, general paralysis of the insane, bulbar paralysis, and various other forms of paresis, often come to my clinique complaining simply of weakness. Cases of *malingering* offer far greater difficulty in diagnosis from general debility, for in both cases we are almost entirely dependent upon the patient's own statements. The question of motive should be considered and an exhaustive examination made by the most up-to-date scientific apparatus, but even then we may in justice be compelled to give the patient the benefit of the doubt. My experience at the Paddington Infirmary taught me in many cases that it is only by keeping the patient under daily observation, and with the aid of intelligent, experienced, and well-trained nurses, that a correct conclusion can be gained. I have no doubt that large numbers of able-bodied malingerers gain admission to the infirmaries in Great Britain as cases of general debility. *Hysteria* and *neurasthenia* may require to be distinguished from debility. The *causes* of debility are discussed in §§ 402 and 419.

Pallor of the Skin, *i.e.*, deficiency of its normal colour, is a frequent accompaniment of cases in which debility is complained of by the patient, and the experienced observer can detect considerable variations in the different varieties of pallor belonging to several diseases which will be alluded to shortly (§ 402 *et seq.*).

Fallacies. Slight *jaundice* may resemble some forms of pallor, but in jaundice the sclerotics are unmistakably tinged and the urine may contain bile pigment. In *town-dwellers* who suffer from a deficiency both of fresh air and sunlight pallor of the face is common, and in certain "delicate" families a pale face is more or less normal. Europeans who have lived long in the *tropics* are habitually pale and "anæmic" looking, but the blood may not reveal any changes of anæmia. On the other hand chlorotic girls may occasionally present flushing of the face and redness of the lips though undoubtedly suffering from anæmia.

Emaciation or loss of flesh may also be associated with general debility, and its presence adds considerably to the gravity of a case, for it indicates either serious organic disease such as cancer or tubercle, or definite defect in the alimentation or metabolism of the body, such as intestinal trouble or chronic Bright's disease. It is manifested to the patient by his clothes becoming looser, or his face becoming thinner, or to the physician by pinching up a fold of skin between the finger and thumb. But the only reliable test is a definite loss of weight, and it is advisable at the outset to ascertain and record the weight of all patients who come to us complaining of debility. To ascertain the net weight, $\frac{1}{20}$th of the gross weight may be deducted for summer, and about $\frac{1}{15}$th for winter clothes. Every consulting room should be provided with scales. The causes of emaciation are discussed in § 416 *et seq.*

Fallacies. A normal loss of adipose tissue may occur about the climacteric, but the reverse is quite as usual. In advancing years loss of flesh, or the reverse, is normal in some families: both are largely a question of heredity. Amyotrophy unless generalised is not apt to be confused with emaciation; it is usually more or less localised. The diet a person has been taking will within certain limits influence his weight considerably, and one who has been taking only nitrogenous food (*e.g.*, the so-called Salisbury diet) may be many pounds under his normal weight.

PART B. PHYSICAL EXAMINATION.

§ **395.** The physical examination of cases of general debility, pallor, or emaciation comprises: -1st, EXAMINATION OF THE VISCERA; 2ndly, OBSERVATIONS ON THE WEIGHT, and in some cases on the TEMPERATURE; and 3rdly, AN EXAMINATION OF THE BLOOD.

1. An examination of the VISCERA should be very systematically and thoroughly conducted (see scheme, pp. 8 and 9), because we may be dealing with some incipient disease the signs of which are obscure. Enquiries should be especially directed to the state of the digestive organs, and the urine should be carefully examined. Special importance will attach to the latter when we know more about the causes and consequences of abnormal haemolysis (blood destruction).

2. The WEIGHT of the patient should be noted and if possible compared with previous records. It may be desirable also to take the patient's TEMPERATURE if any pyrexia be suspected, and to obtain a series of records, § 348, p. 583.

3. An examination of the BLOOD is generally necessary, especially in cases where any form of anaemia is suspected. This in its complete form consists of (1) estimation of the hæmoglobin; (2) blood counts of the red and white corpuscles; (3) examination of blood films. In most cases these three will be sufficient for a routine examination; but in other cases it is necessary to make (4) an examination for parasites and other abnormal constituents; and (5) certain physical and chemical properties of the blood.

EXAMINATION OF THE BLOOD.

§ **396. Apparatus** and **Methods.** APPARATUS REQUIRED. The cover-glasses used in blood examination must be perfectly clean and free

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from greasiness, and for blood films they must be specially thin. Cover-slips should be cleaned in the following manner. (i.) Drop the cover-slips one by one in an enamelled iron pot containing 10 per cent. solution of chromic acid, and boil gently for twenty minutes. (ii.) Turn the slips into a flat glass dish and wash in running tap water until all trace of yellow colour has disappeared. (iii.) Wash in rectified spirit, then transfer them by means of a clean forceps to a small beaker of absolute alcohol. (iv.) Draw off the alcohol and transfer the slips by the forceps to a wide-mouthed glass pot containing absolute alcohol in which they are to be stored. After once being placed in chromic acid the slips must on no account be touched by the fingers. They always require delicate manipulation; a pair of spring forceps which will lie down horizontally is useful for holding them. Cover-glasses should never be laid flat on the table, but have one edge on the table, the other edge leaning on some object. Glass slides require to be cleansed from grease. Alcohol and ether are necessary for cleansing and disinfecting the skin; my own practice is to use A.C.E. mixture.

For the *estimation of hæmoglobin* the following apparatus are in use—the Tallqvist scale, Gowers' hæmoglobinometer, Von Fleischl's hæmometer, Haldane's and Oliver's hæmoglobinometers; Gowers' instrument is the one mostly used. For *counting the blood cells* the Thoma-Zeiss or the Gowers' hæmocytometer is employed. Diluting solutions are required for this purpose. For counting the red cells a solution of sulphate of soda of specific gravity 1025 may be used, or a mixture of sod. sulph. gr. 104, acetic acid ʒj, aq. destill. ℥vj. For counting the white cells a 0·3 per cent. solution of acetic acid coloured by methyl green is used, or Toison's fluid (methyl violet 0·025 grm.: neutral glycerine 30·0 c.c., distilled water 80·0 c.c. Add to this a solution of sodium chloride 1·0 grm., sodium sulphate 8·0 grm., distilled water 80·0 c.c., and filter). The instruments must be carefully cleaned before being put away, first with water, then with alcohol or ether, then with water again.

For *staining the blood* the following stains are those commonly used:—(1) Eosin, grm. xv., abs. alcohol 70·0 c.c., aq. destill. 30·0 c.c. (2) Lœffler's methylen blue—conc. alcoholic soln. of meth. blue, 1 c.c., caustic potash in 0·01 per cent. aqueous solution, 3 c.c. (3) Ehrlich's triple or triacid stain can be obtained from Grübler & Co. of Leipsic in the form of a powder (the Ehrlich-Heidenhain powder) and is mixed thus:—Powder, gr. xv., abs. alcohol 1 c.c., aq. destill. 6 c.c. The powder contains orange G., acid fuchsin and methyl green. (4) Jenner's stain is a specially prepared mixture of eosin and methylen blue in methyl alcohol. (5) For parasitic and other abnormal constituents in the blood met with abroad, Leishman's[1] or Romanowsky's[2] specially prepared stains are employed.

METHOD OF OBTAINING BLOOD. Certain precautions are necessary to obtain satisfactory results in procuring a specimen of blood for examination. A series of observations on the same patient should be carried out as far as possible always under the same conditions, because there are physiological alterations in the constituents of the blood after meals, cold baths and exercise. Blood can be obtained from the back of the finger just above the nail, a handkerchief or string being tied round the selected finger in order to render it hyperæmic. This process is painful and the proportion of corpuscles to the serum is slightly altered by the artificially induced hyperæmia. It is better to obtain the blood from the lobe of the ear. Cleanse the lobe with soap and water, then with ether, and in cleansing render it hyperæmic by rubbing. The skin is then lightly and rapidly pricked. For this purpose a guarded two-edged lancet is the best instrument, but a needle or vaccination lancet may be employed. The lancet should be rendered sterile by wiping with ether, not heated, as that blunts the point. The blood must not be squeezed out, or it will be diluted with lymph from the tissues.

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[1] Leishman's stain is prepared by Grübler, of Leipsic. The method of preparation is described in the *Brit. Med. Journal*, 1901, vol. ii., p. 757.

[2] Romanowsky's stain is fully described in the *Brit. Med. Journal*, 1901, vol. i., p. 635.

If it is required to take away sufficient blood (say up to ℥ss) to examine in the laboratory, for bacteriological examination or for a Widal reaction, a pipette can easily be made in the following way. Take a clean piece of glass tubing about 6 or 8 inches long, sterilise it by heat, and hold the middle in the flame of a Bunsen burner until it is thoroughly soft. Pull the two ends apart *in a straight line* at the moment of taking it out of the flame, thus reducing the middle third of the tube to a capillary diameter. Break it in the middle into two, seal the two capillary ends in the flame, and plug the other ends with tiny plugs of sterilised cotton wool. If properly baked wool is not handy it may be roughly sterilised in the flame. When these pipettes are cold the blood is sucked up through the capillary end (previously broken off afresh), by placing the other end in the mouth, the wool being still left *in situ*. The capillary end is again sealed, and the blood thus obtained can be taken away to the laboratory for examination; but the pipette must be kept upright all the while. It must be remembered also that the clot will separate out, leaving a layer of clear serum above and around it. Another plan of making blood pipettes, used by Professor A. E. Wright, is to heat and pull out the other end of the glass tube also to a capillary diameter, then gently warm this end and turn it up into a short limb of a U-shaped pipette. When required, break off the tips of both ends, and the blood will find its way by capillary attraction through the longer limb into the bulb, which should only be half filled with blood. Seal the two ends in the flame.

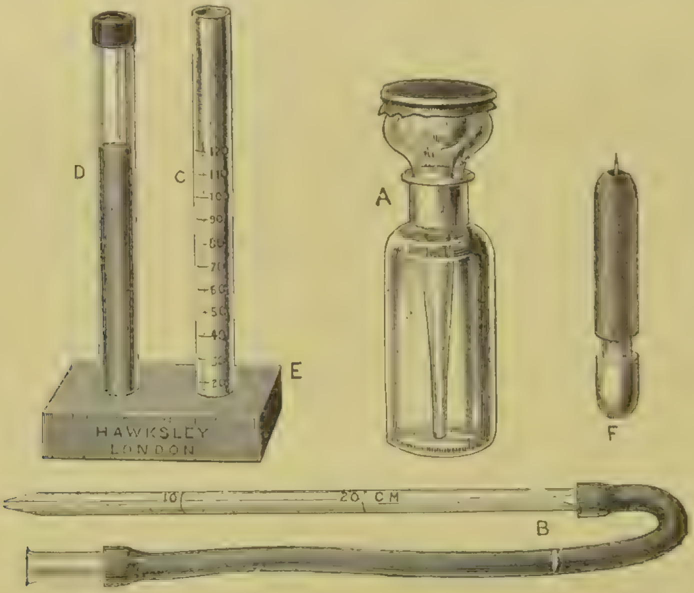

Fig. 111.—Gowers' Hæmoglobinometer.

§ 397. Estimation of the Hæmoglobin. In estimating the quality of the blood the number of red cells is of less importance than the amount of hæmoglobin, their active constituent. Hæmoglobin may be roughly estimated by the *Tallqvist scale*,[1] which consists of a lithographed scale of tints. A drop of blood is sucked up by one of the pieces of blotting-paper supplied, and compared with the scale of tints as soon as the stain has lost its humid gloss. The figures beside the tints represent the percentage of hæmoglobin present, normal being 100. The estimation can only be performed in full daylight.

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[1] The Tallqvist scale can be obtained from Messrs. Allen & Hanbury, Wigmore Street, London, the authorised agents for England.

Another method of estimating the hæmoglobin is by means of *Gowers' hæmoglobinometer*, which consists of the following apparatus (see Fig. 111):—(A) A pipette dropping-bottle with rubber top; (B) a capillary pipette marked at 20 cmm.; (C) an open tube of the same size as D, graduated so that 100 degrees equal 20 cmm. of blood diluted 100 times; (D) a closed tube with a solution of picrocarmine glycerine, the colour of which corresponds to that of normal blood diluted 100 times; (F) a guarded lancet. Place a few drops of distilled water in the graduated tube; with the pipette suck up 20 cmm. of blood and blow this into the tube, mixing to prevent coagulation. To ensure the removal of all the blood from the pipette refill it with water several times and blow into the tube. Hold the tubes together against the light or a sheet of paper, and add water from the dropping-bottle until the colours of both tubes correspond. Good daylight is required for this test. Note the number at the surface-level of mixture. This gives the percentage of hæmoglobin in the examined blood compared to the normal. The amount of hæmoglobin may also be estimated by Von Fleischl's hæmometer or by Oliver's hæmoglobinometer.

Significance of diminution or increase of Hæmoglobin. The amount of hæmoglobin is always expressed in terms of a percentage of the normal standard. Thus, 87 indicates that the amount is 87 as compared with the normal of 100. A diminution of hæmoglobin is the essential feature of all anæmias, but the various forms of anæmia differ as regards the number of blood cells. Thus in chlorosis there is a marked diminution of hæmoglobin in each corpuscle, though their number may not be much diminished. On the other hand, in pernicious anæmia the diminution of hæmoglobin is due to the marked diminution in the number of red cells, each of which contains the normal or above the normal hæmoglobin value. The amount of hæmoglobin in each corpuscle is expressed by making a fraction, the numerator of which is the percentage of hæmoglobin present (as estimated by the hæmoglobinometer) and the denominator of which is the percentage of corpuscles (as estimated by the hæmocytometer). For example:—If the examined blood has 40 per cent. hæmoglobin and 80 per cent. red corpuscles, the value of hæmoglobin in each corpuscle is $\frac{40}{80}$ths normal. This fraction expresses the *colour index* of the blood, or ratio between the percentage of hæmoglobin and the percentage of red corpuscles.

§ 398. **Blood Counts.** An estimation of the number of corpuscles in the blood is in many cases of extreme importance both for the diagnosis and the treatment of disease. Two instruments are in use for this purpose, the Thoma-Zeiss and Gowers' hæmocytometers. The former is more convenient, requiring a smaller quantity of blood, and having a smaller percentage of error. We shall consider first the method of estimation of the NUMBER OF RED CELLS in a cubic millimetre of blood, by means of the *Thoma-Zeiss hæmocytometer*. The apparatus consists of a mixing pipette B, a graduated counting slide A (Fig. 112), and a diluting fluid consisting of sod. sulph. in solution, of specific gravity 1025; or, sod. sulph. gr. 104, acetic acid ʒj, aq. destill. ℥vj. Before starting, see that all the instruments are clean and at hand. Suck up the blood from the drop to mark 0·5 (or 1·0) into the capillary pipette; if any of the blood reach the mixing chamber E the instrument must be cleansed and the process started again, as it is necessary to be very precise in this measurement. Wipe the end of the pipette, plunge it into the diluting fluid and suck it up to the mark 101. Holding the pipette between the finger and thumb, rotate and shake it so as to thoroughly mix the fluids in the mixing chamber: the glass ball in E greatly facilitates this process. Next blow out and discard the clear fluid from the capillary end of the tube and also 3 or 4 drops of diluted blood; then let a fraction of a drop fall upon the graduated platform in the centre of the slide. The drop must be of such a size that when the cover-glass is applied the blood will not run over the edge into the trench around. Place the cover-glass on the drop of blood, and if the cover-glass has been properly cleansed it lies so closely on the outer rim that Newton's concentric colour rings can be observed on the cover-glass. Set the slide aside for a few minutes to allow the corpuscles to settle.

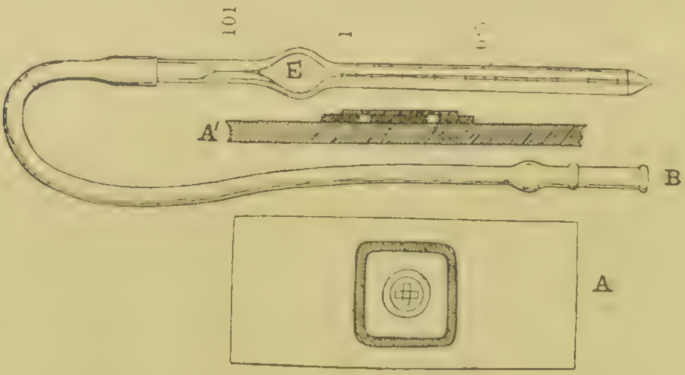

Fig. 112.—The THOMA-ZEISS HÆMOCYTOMETER.

The platform is ruled by cross lines, each enclosing a space of $\frac{1}{400}$ of a square millimetre; the depth of each square with the cover-glass on is $\frac{1}{10}$ of a millimetre. The squares are marked out into sets of 16 by double lines. Count the red cells in three of such sets, that is, in 48 squares. If the corpuscles lie *upon* the lines count those on the upper and left side lines only. Having counted 48 squares, divide the total by 48 to get the average per square. The dimensions of a square are $\frac{1}{400} \times \frac{1}{10} = \frac{1}{4000}$ of a cmm. If the blood was drawn up to mark 0·5 it was diluted 200 times. Let N equal the average number of corpuscles per square, then the number of corpuscles in a cmm. of undiluted blood will $= N \times 4000 \times 200$. If the blood was drawn up to mark I it was diluted 100 times, and the last number will be 100 instead of 200. Example:—Supposing that after counting 48 squares the average is found to be 6 corpuscles per square, the number of corpuscles in 1 cmm. will then $= 6 \times 4000 \times 200 = 4{,}800{,}000$, which is about the average for a healthy person.

Gowers' Hæmocytometer (Fig. 113) consists of (A) a pipette graduated for 995 cmm.; (B) a capillary tube graduated for 5 cmm.; (C) a counting slide; (D) a mixing vessel; (E) a stirrer; and (F) a guarded lancet.

Method. Suck up 995 cmm. of the diluting fluid (*vide supra*) and blow it into the mixing vessel. Blow 5 cmm. of blood into this and thoroughly mix. The blood is now diluted 200 times. Place a drop of the diluted blood on the graduated slide; lay a cover-glass over the slide and fix it by clips. Each square on the slide has an area of $\frac{1}{100}$ mm., and its depth is $\frac{1}{5}$ mm. Count the corpuscles in several squares (say 10) and take the average. The resulting number multiplied by 100,000 gives the number of corpuscles in 1 cmm. of undiluted blood ($\frac{1}{500}$ cmm. = the size of each square, therefore $\frac{1}{500} \times 200$, the amount of dilution, = 100,000).

Significance of diminution or increase of Red Cells. In health the average number of red cells per cmm. is about 5 millions in the male and 4½ millions in the female. It is diminished with menstruation, childbirth, and by drinking much fluid. It may be increased to 7 or 8 millions in newly-born children, in plethoric persons, after sweating and fasting, and in those living at high altitudes. *In disease* the number is diminished slightly in all forms of secondary anæmia, though it may be normal in chlorosis. Great diminution in number—*i.e.*, under 3,000,000—occurs after severe hæmorrhages, acute septicæmia, malaria, and severe secondary anæmia. In leukæmia there may be only 2 millions per cmm., and in pernicious anæmia they may be as low as half a million. In disease there is very rarely an absolute increase in the number of red cells, but there is a relative increase when a large quantity of fluid is lost from the body, owing to the concentration of the blood, *e.g.*, in the algide stage of Asiatic cholera (in which the number may reach 6½ millions per cmm.), after the removal of a large quantity of ascitic fluid, and after persistent vomiting. In cyanosis and phosphorous poisoning also there is an apparent increase in the number of red cells.

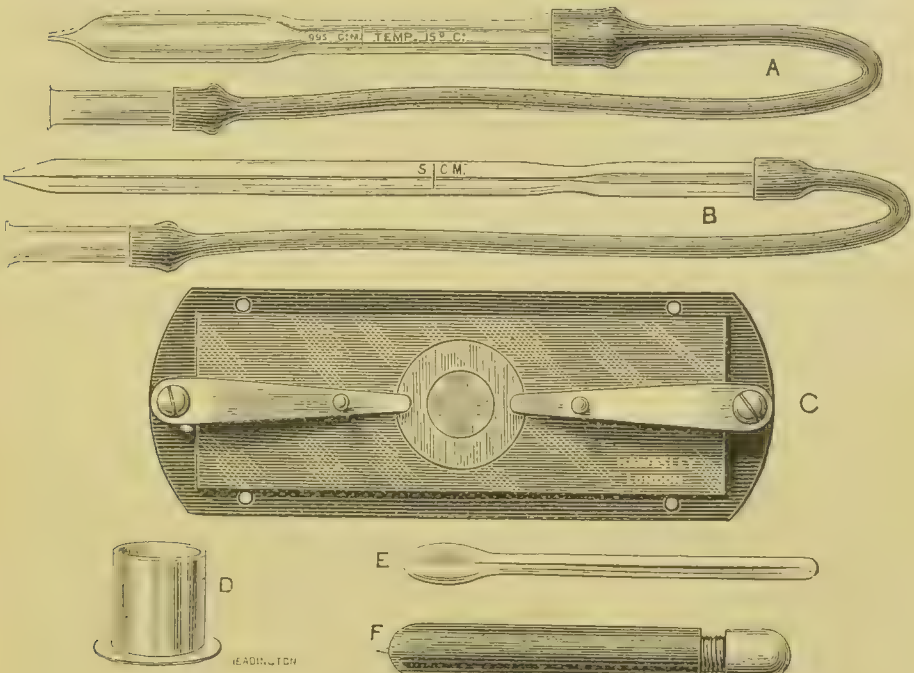

Fig. 113.—Gowers' hæmocytometer.

The number of leucocytes per cmm. and the proportion of white to red will next be considered. To enumerate the leucocytes the Thoma-Zeiss instrument is most convenient, and a special pipette is provided which gives a dilution of 1 in 20 (if the blood be drawn up to mark 0·5); or 1 in 10 (if up to mark 1). The leucocyte pipette is recognised by its having a mark 11 at the top of the mixing chamber, and the diluting fluid is sucked up to this mark. As a diluting fluid simple normal saline solution may be used, or a 0·3 per cent. solution of acetic acid coloured by methyl green. The acetic acid dissolves the red cells and the green stains the nuclei of the white. A still more useful diluting fluid is that of Toison (*vide* § 396), which does not destroy the red cells, but stains and thus differentiates the leucocytes. The calculation is carried out by counting the number of leucocytes in all the 256 squares. If the blood was diluted 20 times and 40 leucocytes are found, then $\frac{40}{256} \times 4000 \times 20 = 9200$ per cmm., which is a high normal. Gowers' hæmocytometer can also be used and then the number of leucocytes in 50 squares should be counted and the resulting total multiplied by 2,000 to obtain the number in 1 cmm. of blood.

Significance of increase or diminution of the Leucocytes. In health the normal number of leucocytes is 7,000 per cmm. (*i.e.*, 1 white to 700 red cells). On a normal field, with $\frac{1}{6}$ English objective, and a No. 2 eyepiece, about three or four white cells are generally seen. In the newly-born there may be over 17,000 and in early childhood from 10,000 to 14,000 leucocytes per cmm. During pregnancy, after meals, cold baths and exercise there is an increase in the number of leucocytes. The polynuclear neutrophil leucocytosis (§ 399) is the most usual form of leucocytosis both in health and disease. *In disease* the number of leucocytes is INCREASED (leucocytosis) in the following conditions, these being the chief conditions in which leucocytosis is of use for diagnosis or prognosis :—

Abscess and suppuration,
Septicæmia,
Pneumonia,
Erysipelas,
Scarlet fever,
Osteomyelitis,
Malignant endocarditis,
Tuberculous meningitis,
} in all of which the leucocytosis is due to increase of the polynuclear neutrophil cells.
In splenic leukæmia there is an increase of myelocytes,
In lymphatic leukæmia and chloroma an increase of lymphocytes,
In anæmia splenica infantum an increase of myelocytes,
In some skin diseases an increase of eosinophil cells, and
In lymphosarcoma a slight increase of lymphocytes.

A DIMINUTION of the number of leucocytes (leucopenia) is rare: it occurs in splenic anæmia, enteric fever and measles.

Staining and *blood films are necessary* for the estimation of the number of *different kinds* of leucocytes (the differential count), and this will now be described.

§ 399. **Microscopic examination** of blood and **Blood films.** Alterations in the shape and size of the blood cells may be seen by examination of fresh blood films, but for accurate examination of the structure of the red and white cells and a differential count of the leucocytes it is essential that blood films be fixed and stained. Blood may be obtained from the lobe of the ear by the method above described (§ 396). A microscopic examination of fresh blood may be made by applying a clean slide lightly to the drop of blood, placing a cover-glass on it, and examining under the microscope. For this method any good microscope will do with a $\frac{1}{4}$ or a $\frac{1}{6}$-inch English objective (or a Zeiss's D) and a No. 2 eyepiece; but for the differential examination of leucocytes and for bacteria a $\frac{1}{12}$-inch oil immersion lens is necessary. It is a great advantage to have a nose-piece on the microscope capable of carrying two or three objectives, so that one can first examine the specimen with a low, and then with a high power. It is well to make oneself familiar with the changes the blood undergoes in a short time after such a method of preparation. If it be desired to preserve such a specimen for some hours, ring the edge of the cover-glass with vaseline to prevent the entrance of air. In this simple way we are able to note any abnormality in the shape of the red cells, or the presence of abnormal constituents, such as particles of pigment, filaria sanguinis hominis, or the spirillum of relapsing fever. We may also note any excess of white cells, for in an ordinary ¼-inch field only 2 to 4 white cells are generally visible. Rouleaux formation is also noted in normal fresh blood, *i.e.*, the red cells run together, leaving clear the concave spaces in which blood platelets are seen. The white corpuscles are spherical, clear and nucleated.

A film may be made upon a cover-glass or a slide; for ordinary purposes the slide method is the easier. It is essential that the slides or cover-glass be absolutely clean and free from greasiness. Lay the surface of the slide lightly on the drop of blood exuding from the lobe of the ear, and with the smooth edge of another slide or with a morsel of tissue paper spread out the blood in a *thin* film by pushing the drop along the surface. Care must be taken not to handle the slides too much or to breathe upon them. For those who have the necessary manipulative skill the cover-glass method gives the best results. Two cover-glasses, preferably of oblong shape, are required; place one upon a piece of white blotting-paper: hold the second in the forceps, and touch the summit of the drop of blood with the half of the slide furthest from the operator, so that a little blood adheres to the glass. Lay cover-glass No. 1 on cover-glass No. 2 so that their ends overlap by half of their length. Let the blood spread out evenly between the two into a thin layer; take each cover-glass edgewise between the thumb and forefinger of each hand and slide them quickly apart in their long axes. If successfully done each glass is covered with a *thin*, uniform layer of blood, which quickly dries, and can be taken away for fixing and staining on the same or the subsequent 2 or 3 days.

To *fix a blood film* is a necessary preliminary to staining and mounting. If not performed correctly the corpuscles will shrivel up or break, and will not take the stains properly. Fixing may be performed in several ways:—(i.) by immersing the film in a mixture of absolute alcohol and ether for a quarter of an hour and allowing to dry; (ii.) by immersing in a saturated solution of corrosive sublimate for one to three minutes and then washing very thoroughly in water; or (iii.) fixing by the aid of heat. This last method is rarely employed except when it is desired to stain the film subsequently with the Ehrlich-Biondi stain. Experienced workers obtain good results by passing the film rapidly through the Bunsen flame twenty times, but this plan cannot be recommended to the novice. Another method is by placing the slide or cover-glasses on the centre of a copper plate, one end of which is heated by a Bunsen burner. The heat is regulated by placing a drop of water at the other end; when it steams the plate is too hot.

In the choice of *stains* we employ different dyes according to whether we wish to stain the protoplasm, or the granules of the cells, or the nuclei: the resulting differentiation of the structure and shape of the cells is of extreme importance in the diagnosis of disease. It is usual to stain first with eosin, then with a nuclear stain such as methylen blue. Stains are acid or basic. The common acid stains are eosin, aurantia, and acid fuchsin; the basic stains are methylen blue, hæmatoxylin, and gentian violet. The protoplasm of the red cells takes up acid dyes only;

normal nuclei take up basic stains. The granules met with in the protoplasm of the various leucocytes take up different stains; some have an affinity for acid stains such as eosin, and are known as oxyphil or eosinophil granules; some take up basic stains such as methylen blue and are called basophil granules. The granules occurring in the ordinary polynuclear leucocyte were at one time supposed to take up both acid and basic granules, and hence were named neutrophil granules. It is now known that these granules take up faintly acid stains, though the cell is still named "polynuclear neutrophil" for purposes of description and differentiation. The three methods of staining which are in common use are:—the eosin and methylen blue stain: the eosin and hæmatoxylin stain: and the Ehrlich-Biondi or triple stain. Filter every stain before using.

Eosin and methylen blue method. Place the film in a solution of eosin (½ per cent. in 60 per cent. alcohol) for 2 minutes; wash by holding the glass under a gentle stream of water running from a tap; dry by blotting-paper and then pass rapidly once through a Bunsen flame, taking care that the film does not become hot: place in a solution of methylen blue for 1 or 2 minutes; wash and dry as before; mount in Canada balsam. The red corpuscles are stained pink, the nuclei of the leucocytes blue, the granules of the polynuclear cells show as small faint pink granules; the eosinophil cells show coarse red granules, degenerate red cells take up some of the basic (blue) stain also. The protoplasm of the leucocytes usually remains unstained, but in some of the mononuclear cells fine blue granules are seen at the periphery of the cell; in others the protoplasm is diffusely stained a pale blue without granules. The mast cells show coarse granules of a violet blue. For the *eosin and hæmotoxylin* method the same steps are taken as in the eosin and methylen blue method: Delafield's hæmotoxylin being used instead of the latter (Plate IV.).

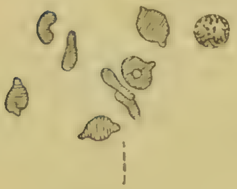
Fig. 114.—Poikilocytosis.

The Ehrlich-Biondi stain requires films which have been *fixed by heat.* For accurate results by heat-fixing a small copper oven from which a thermometer projects is almost indispensable. The cover-glasses or slides are wrapped in paper and placed inside the oven: a Bunsen burner is placed 10 to 12 inches below. The films are then baked 1 to 2 hours at a temperature of 120° C., or 10 to 15 minutes at a temperature of 130° C. It is useful for the differentiation of the leucocytes and the detection of degenerative changes in the red cells. Stain from 1 to 6 minutes, wash thoroughly, dry and mount. If the film has been properly prepared and heated the red cells appear bright orange yellow; if underheated they look brown; if overheated, pale yellow. The neutrophil granules show a pink or pale violet hue; the eosinophil granules are copper; the nuclei of the leucocytes are a greenish blue; the nuclei of the nucleated red cells green or black (Plate IV.). As the triple stain is not a basic stain none of the mononuclear cells show granules.

Bremer found that DIABETIC BLOOD has a special reaction with certain stains. Heat the diabetic blood film together with a control film of normal blood at 135° C. in the oven for 6 to 10 minutes. Stain 2 minutes in 1 per cent. solution of Congo red. Normal blood is stained red; diabetic blood is not stained.

Variations of the red blood corpuscles in disease may consist of (1) variability in form (poikilocytosis): (2) variability in size:

Plate IV

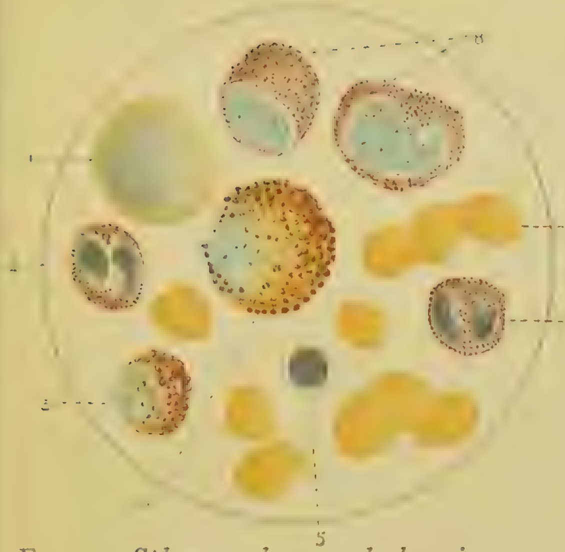

FIG. 1.—*Spleno-myelogenous leukaemia.*
(*Obj. oil immers.* $\frac{1}{12}$—*Eyepiece* 3.)

1, Large mononuclear leucocyte; 2 and 6, polynuclear leucocytes; 3, eosinophil leucocyte; 4, eosinophilic myelocyte; 5, lymphocyte; 7, red corpuscles; 8, myelocytes.

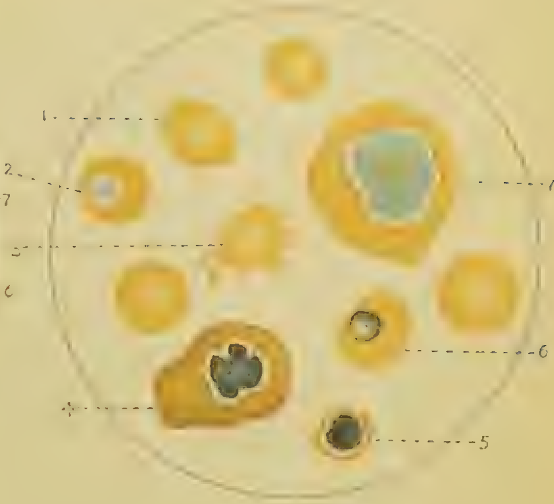

FIG. 2.—*Pernicious anaemia.*
(*Obj. oil immers.* $\frac{1}{12}$—*Eyepiece* 3.)

1, Normal red cell; 2 and 6, nucleated red cells, normoblasts; 3, cell with ragged protoplasm; 4, old megaloblast with dividing necleus; 5, microblast; 7, megaloblast.

Figs. 1 and 2 are stained by the *Ehrlich-Biondi* method; this shows the copper colour of the coarse eosinophil granules, and the lilac colour of the fine neutrophil granules which occur in polynuclear leucocytes and myelocytes. Red corpuscles stain a yellowish brown.

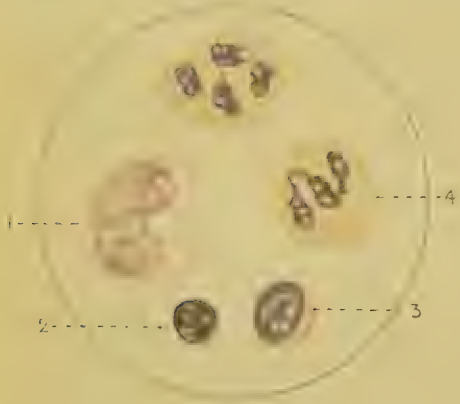

FIG. 3.—*Figure to show varieties of leucocytes.*
(*Obj. oil immers.* $\frac{1}{12}$—*Eyepiece* 3.)

1, Large mononuclear leucocyte; 2 and 3, lymphocytes; 4, polynuclear leucocytes.

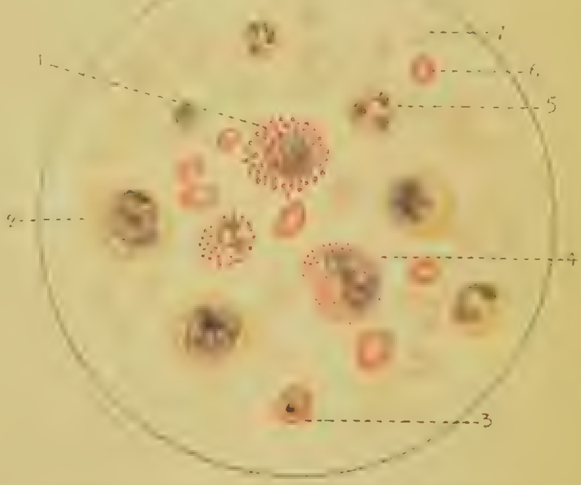

FIG. 4.—*Spleno-myelogenous leukaemia.*
(*Obj.* $\frac{1}{8}$—*Eyepiece* 2.)

1, Eosinophilic myelocyte (coarse granules); 2, myelocyte; 3, nucleated red corpuscle; 4, myelocyte with fine eosinophil (oxyphil) granules, intermediate between ordinary myelocytes and eosinophilic myelocytes; 5, polynuclear leucocyte; 6, normal red corpuscle; 7, microcyte (small red cell).

Figs. 3 and 4 are stained by the *Eosin* and *Haematoxylin* method. Nuclei are stained blue; protoplasm pink; eosinophil granules, red. Note the increased number of myelocytes. The neutrophil granules of the polynuclear cells and myelocytes are not shown by this method.

[illegible]

(3) nucleation. Normal red cells are circular, bi-concave, non-nucleated discs measuring in size 6 to 7 u or $\frac{1}{3200}$ of an inch.

(1) *Poikilocytosis* (Fig. 114 and Plate IV.) is a variability in the shape of the red cells. They may resemble a flask, a pear, or a kidney. This change used to be regarded as pathognomonic of that serious disease pernicious anæmia; but the change is also found in leukæmia, splenic anæmia of children, and in severe secondary anæmia such as occurs in cancer, nephritis and malaria. With variation in shape the red cells in any form of profound anæmia appear to undergo degenerative changes—clear hyaline spaces (vacuolation) are seen inside the corpuscles when the specimen is examined just after removal from the body. It must be remembered, however, that such spaces as these may also be seen in normal blood about an hour after it leaves the body. Other more definite changes are seen in the staining of the degenerate cells, for whereas normal red cells take up only acid stains, these degenerate cells take up both acid and basic stains, and their substance stains irregularly. This property is known as polychromatophilia.

(2) Variability in *size*. The normal red corpuscle measures about 7 μ; red cells measuring 3 to 5 μ are termed microcytes, and those measuring 9 to 12 μ macrocytes. Both variations are met with in pernicious anæmia, leukæmia, the splenic anæmia of children, and in severe secondary anæmia. They are also seen in severe cases of chlorosis.

(3) *Nucleation*, if present, is made more evident on diluting a drop of fresh blood with a drop of $\frac{3}{4}$ per cent. saline solution coloured with methyl violet. It is also well brought out by the triple stain (*v. supra*, and Plate IV.). Nucleated red cells are found in all cases where there is a great diminution in the number of the red corpuscles, as, for example, in leukæmia, pernicious anæmia, and severe secondary anæmia. They are very rare in chlorosis, but have been seen. These nucleated red cells must be distinguished from lymphocytes, which resemble them approximately in size. The nucleated red cells differ in having a wider rim of protoplasm round the nucleus and the more homogeneous staining of the nucleus. The nuclei may show karyokinesis, or may be degenerate. There are 3 distinct forms of nucleated red corpuscles: the normoblast, about the same size as a normal red cell; the large form or megaloblast, which is about 3 to 5 times the size of an ordinary red cell; and the microblast (or immature red cell), which is smaller than the normal red cell. The presence of the megaloblast in the blood is generally of grave import, indicating serious need for rapid regenerative changes in the blood. These cells are usually polychromatophilic (*vide supra*). They occur in large numbers in pernicious anæmia and in leukæmia.

In *primary anæmias* (p. 725 below) the essential blood change is a diminution of hæmoglobin, accompanied in the majority of instances by a greater or less diminution in the number of red cells, and various degenerative and regenerative changes.

In *secondary anæmia* Dr. Cabot[1] describes 4 stages in the blood alterations. These stages hold good except in anæmia after malaria, hæmorrhages, and acute septicæmia, when the 3rd may precede the 2nd stage. (1) Diminution of the hæmoglobin and lowered specific gravity, without diminution in the number of red cells; (2) diminution in the haemoglobin. loss of rouleaux formation. slight diminution in the number of red cells. with degenerative changes in their shape and size—poikilocytosis. microcytes and macrocytes. vacuolation and polychromatophilia; (3) diminution of the haemoglobin and the number of red cells (under 3,000,000); (4) in addition to the above there are regenerative changes—microblasts, normoblasts. and megaloblasts. It will be observed that differences between primary and secondary anaemias are not at the present time sufficiently characteristic for differentiation by the microscope. but the prognosis and treatment may depend upon the result of microscopic examination.

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[1] "Guide to the Examination of the Blood," by R. Cabot, 1898.

Variations in the Leucocytes may occur in regard to their absolute number (blood count. *vide supra*). their structure. and the relative number of one kind or another (differential count). There are several kinds of leucocytes, and it is possible to identify the cause of an increase in the leucocytes by the predominating variety present. For this purpose and in order to make a differential count to ascertain the relative proportion of the several varieties it is necessary to employ the staining methods given above (*e.g.*, eosin and methylen blue, or Ehrlich-Biondi).

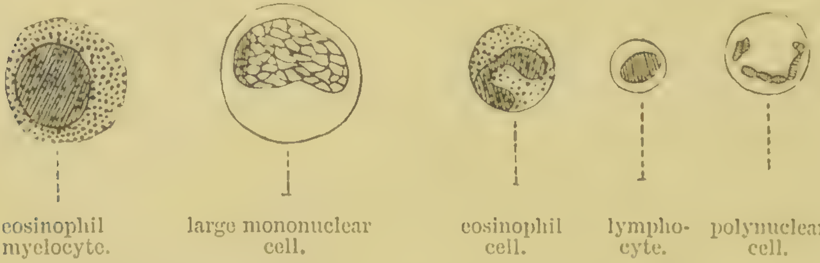

Fig. 115.—NORMAL LEUCOCYTES (see also Plate IV.).

The varieties of leucocytes found *in health* are as follows (Fig. 115):—

In 100 leucocytes:—

Polynuclear	60 to 70
Small mononuclear leucocytes (lymphocytes)	30 to 20
Large mononuclear leucocytes	5
Transitional forms	3
Eosinophil cells	2
	100

(1) In the *polynuclear neutrophil leucocytes*, which form 60 to 75 per cent. of all leucocytes in the blood and have an average diameter of 13·5 μ. the nucleus is long and lobed. giving the appearance of being multipartite. and the protoplasm of the cell contains fine neutrophil granules. The so-called neutrophil granules are really faintly acid; with the Ehrlich-Biondi stain they show fine reddish or lilac granules. This cell originates from the spleen and bone-marrow. and is actively amoeboid and phagocytic (microbe devouring). (2) *Lymphocytes*. 20 to 30 per cent.. are small leucocytes, without granules. with one large nucleus and very small amount of surrounding protoplasm: coming chiefly from the lymphatic glands and adenoid tissue. They are neither amoeboid nor phagocytic. (3) *Large mononuclear* or "hyaline" cells, 4 to 8 per cent., have one large spherical nucleus and a larger amount of protoplasm than the lymphocytes. With the Ehrlich-Biondi stain these cells show no

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granules, but with a basic stain such as methylen blue some of them show a few fine blue granules. These cells are supposed to come from the bone-marrow, and are slightly amœboid. (4) *Eosinophil polynuclear* leucocytes with coarse eosin-staining granules, 2 to 4 per cent., coming from the bone-marrow, are slightly amœboid but not phagocytic. (5) *Basophil polynuclear* leucocytes or mast cells, ½ per cent., some with fine, some with coarse basophil granules seen by prolonged staining with a basic stain. (6) *Transitional forms* between 3 and 1 above are found. Their nuclei are faintly stained, and of a horse-shoe shape.

Variations of Leucocytes in Disease. Variations in the number of the leucocytes in health have been referred to (p. 712), and a list of causes of simple increase in number of leucocytes has been given. Polynuclear neutrophil leucocytosis is the commonest form of increase of the white cells. When leucocytosis is present a differential count is desirable: the disease may often be diagnosed by the predominant variety present. Several hundred leucocytes must be counted, the number of each variety noted on a piece of paper, and the percentage of each calculated. In many cases it may be necessary to make this count daily, *e.g.*, to watch the steady daily increase of polynuclear neutrophil cells, which may denote a perityphlitic or other obscure abscess within the body. Except in leukæmia, leucocytosis rarely passes beyond 100,000 per cmm. (normal about 9,000 per cmm.).

(1) The *polynuclear neutrophil* cells constitute as just mentioned the majority of the white cells in the blood. They are greatly increased in infective diseases, such as pneumonia, septicæmia, erysipelas, scarlet fever, and in local inflammations or abscesses such as appendicitis and osteomyelitis. So true is this that an unfavourable prognosis in croupous pneumonia can be based upon a small degree of leucocytosis. In typhoid fever the onset of a suppurative complication may be diagnosed by the presence of an increased number of polynuclear neutrophil leucocytes. A diagnosis may be made between typhoid fever and tuberculous meningitis from the fact that in meningitis there is leucocytosis, but there is none in typhoid fever uncomplicated by abscesses. Again, so long as there is leucocytosis present in convalescence from any acute inflammation a patient should be kept in bed. If leucocytosis be found, even without definite physical signs pointing to an inflammatory condition, the onset of inflammation can almost with certainty be predicted. In this way blood examination comes to be of the highest importance in the diagnosis of obscure abdominal cases, as a polynuclear increase is rightly regarded in such cases as an indication for operation. The blood signs of deep-seated suppuration are increasing leucocytosis with a high percentage of polynuclear leucocytes, increase of blood platelets, increase in the fibrin, and glycogenic degeneration of white cells, *i.e.*, staining with iodine. In cancer of the stomach it has been found that there is often no increase of leucocytes one hour after meals, as would occur in health or in simple ulcer of the stomach. In all severe stomach trouble leucocytosis after digestion is diminished or absent. After profuse hæmorrhages, in malignant cachexia, gout and chronic renal disease leucocytosis is present. The subcutaneous injection of irritants also produces leucocytosis.

(2) The *increase of eosinophil* cells occurs in one form of leukæmia (see below), in several skin diseases, notably pemphigus and psoriasis. In asthma they may be increased to 25 per cent. or more, and this may be

an aid to its diagnosis from cardiac disease and mediastinal tumour. Local accumulations of eosinophils also occur in the bronchial secretion of asthma and around cancer growths. It is found also in those suffering from trichinosis and ankylostomiasis, indeed in all parasitic diseases (*vide infra*).

(3) *Lymphocytosis* (increase of lymphocytes) occurs in children suffering from gastro-intestinal disorders, rickets and syphilis. In adults it occurs in lymphatic leukæmia (90 per cent.), and less markedly after some infective diseases and secondary syphilis. The diagnosis of Hodgkin's disease from lymphatic leukæmia may depend upon the examination of the blood. In the former there may be no leucocytosis, or if present there is an increase mainly of the polynuclear leucocytes; in the latter the lymphocytes are greatly increased.

(4) *Other forms of leucocytes* which make their appearance in disease are (i.) myelocytes, very large cells, mononuclear, some with fine neutrophil, some with coarse eosinophil granules, coming from the bone-marrow. They are not amœboid or phagocytic. Myelocytes occur in the spleno-myelogenous form of leukæmia in large numbers (30 per cent.), and in anæmia splenica infantum. They occur in smaller numbers in bone-marrow sarcoma and severe anæmia after hæmorrhage. (ii.) Pseudo-lymphocytes of various sizes, with fine neutrophil granules, may be seen in similar cases, but are rare.

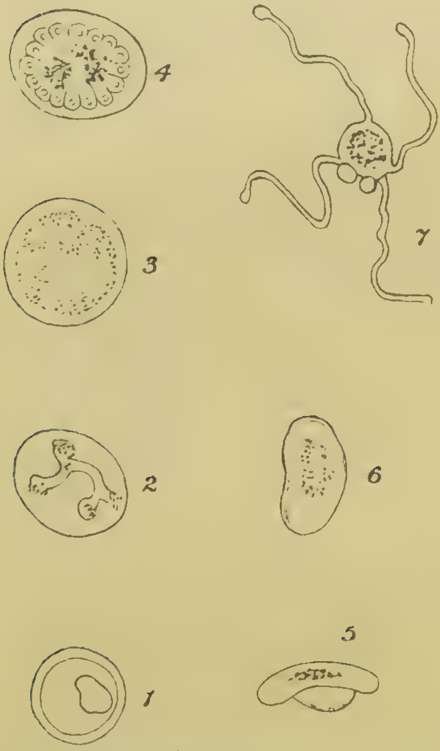

Fig. 116.—Parasite of Malarial fever. 1 to 4 = stages in benign tertian; 5 = crescent body; 6 = oval body; 7 = flagellated body.

Melanæmia is a term applied to certain pigment granules in the blood which occur after ague, relapsing fever, and some melanotic tumours. They appear either in minute black lumps or are enclosed within the cells. They are probably disintegrated blood pigment.

Blood Platelets. As yet no clinical importance has been attached to the blood platelets. They are frequently not observed in the ordinary methods of blood examination. In order to find them a cover-glass must be placed on a slide, with the edges of both corresponding. Place the edges near the forming drop of blood, which by capillary attraction will immediately spread out into a film. Stain with equal parts of normal saline solution and glycerine saturated with dahlia. The blood platelets are seen with a $\frac{1}{12}$-inch oil immersion lens as irregular bodies, small, apt to run together in clumps. They number about 300,000 per cmm. These bodies are increased in leukæmia, grave anæmias and pyogenic conditions, and diminished in purpura, hæmophilia, typhus, erysipelas and malaria.

Blood Dust is a term which has been given to a number of small, clear bodies in the blood, with a vibratile motion, about $\frac{1}{2}$ to 1 μ in size. They are supposed to be granules extruded from neutrophil and eosinophil cells.

§ 400. Parasites found in the Blood. The MICRO-ORGANISMS which can be detected in the blood (such as glanders, anthrax, septic microbes, the spirillum of relapsing fever, the typhoid and influenza bacillus), and their methods of detection, as far as it is applicable to clinical work, are referred to in Chapter XX.

The chief PARASITES which have been found in the blood are the filaria sanguinis hominis, distoma hæmotobium, the malaria parasite, the protozoa of Kala-Azar, and the trypanosoma.

The PARASITE OF MALARIAL FEVER is a protozoon, inhabiting the red corpuscles, which it destroys, but it does not invade other tissues. There are three well-marked varieties of the parasite, distinguished from each other by their intracorpuscular development, and these varieties correspond to the three types of malaria known as benign tertian, quartan, and malignant tertian fever. The life-history of the protozoon runs through two stages: (i.) the asexual or intracorpuscular stage in man, and (ii.) the sexual form within the body of a mosquito belonging to the genus anopheles (Fig. 102, p. 638). The tertian parasite which gives rise to the *benign* form of malaria is the least virulent (Fig. 116). It is first seen within the corpuscle as a small, clear, ovoid body about 2 μ in diameter, possessing active amœboid movement. It gradually increases in size, and after the lapse of a few hours becomes ring shaped, with very finely granulated pigment collecting about its centre. In the benign tertian form there is at the same time marked hypertrophy of the corpuscle, with a striking decrease in its colour. At the stage of full growth the parasite occupies nearly the whole of the enlarged corpuscle, and now it may follow either of two lines of development: (i.) the pigment gathers as a solid mass in the centre of the parasite, the protoplasm divides by a radial arrangement into well-marked rosettes of from 15 to 20 segments; these disintegrate as spores, and masses of insoluble pigment enter the blood stream. This phase is known as "segmentation"; it is complete in about 48 hours, and corresponds clinically to a fresh paroxysm of the fever. The intracorpuscular development just described is asexual. (ii.) The sexual development takes place in the female mosquito, which sucks the blood of a malarial patient, and takes into its stomach the protozoon in the phases above mentioned. All die except the male and female gametocyte (which are crescentic in the malignant tertian type), the female being distinguished by the pigment round the nucleus. The male gametocyte develops within the mosquito into a flagellated body, and one of the flagella unites with the female gametocyte to form an oval-shaped body with pointed ends. This projects from the stomach wall of the mosquito into its body cavity, and becoming spherical it subsequently divides into numerous cells and these again into curved, needle-shaped bodies. These are carried by means of the salivary gland of the mosquito into the blood of the person bitten, and so into his red blood corpuscles.

The parasite of the *malignant tertian* fever is first seen in the red blood cells as a tiny, unpigmented, hyaline body, 48 hours being needed for its development. At first it exhibits energetic amœboid movements, but ultimately settles into a bright, colourless, ring-like form, with one or two pigment granules contained therein. There is frequently multiple infection of the red corpuscles, which at first hypertrophy, then shrink, and become a brassy hue. The rosette or sporulating stage is rarely seen. In about a week (during the period of remission) characteristic crescent bodies, containing masses of coarse pigment granules, begin to appear, and increase in number rapidly. They are incapable of sporulation, and represent the sexual form—the gametocyte.

The *quartan* parasite, the easiest form for the beginner to study because of its visibility, first appears as a small, round, clear speck, resembling a vacuole but with feeble amœboid movement. It takes from 60 to 72 hours to complete its cycle. By the third day, pigment, coarser and blacker than that of the tertian form, gathers round its periphery. On the 4th day segmentation takes place, the pigment flows in towards the centre, and here forms the radiating lines which produce the beautiful "daisy rosette" so characteristic of the quartan parasite. It breaks up eventually into 8 to 10 spores, and these with the insoluble pigment become free in the blood stream. The development of the gametocyte resembles that of the benign tertian variety. There is no hypertrophy of the red corpuscle.

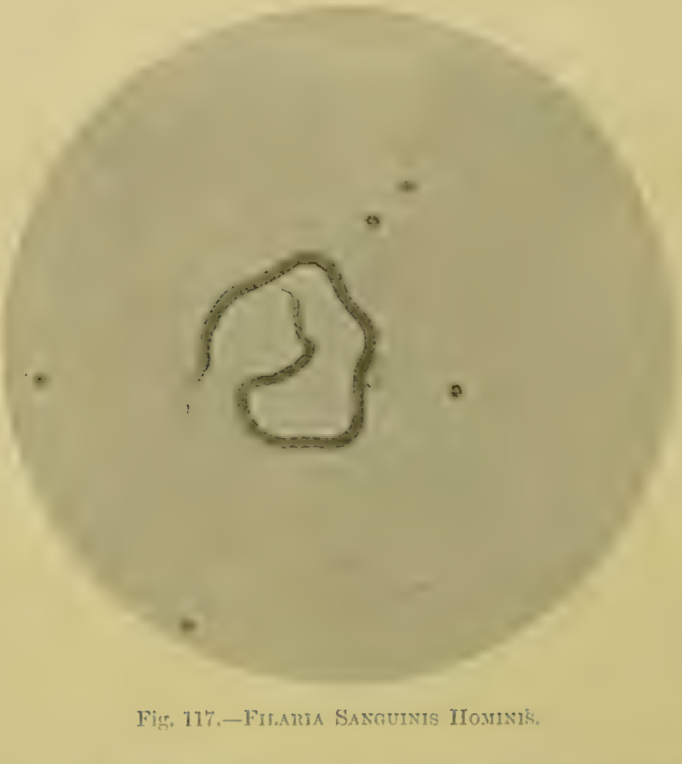

Fig. 117.—Filaria Sanguinis Hominis.

STAINING is not absolutely necessary for the detection of the parasite, which appears as a clear ovoid body within the red cell. Sir Patrick Manson's rules should be followed. It is best to examine the blood unstained at first, just before or during a rigor. *Method*—the first drop or two of blood must be wiped away, and a clean cover-glass applied to the summit of the next drop, then lightly placed on a slide. The blood should then show areas with several zones; if these zones do not appear it is simply waste of time to examine the specimen. To prevent evaporation and crenation it is well to ring the glass with vaseline. The zones are :—

(1) a central or empty zone; (2) a zone of isolated or scattered corpuscles; (3) a third zone in which the corpuscles lie closely together, the "single layer" zone; (4) beyond this the corpuscles lie upon each other, the "heaped up" zone. Look for the parasite in the "single layer" zone. To stain, employ the eosin methylen blue method (p. 714). The red corpuscles are stained light red, the nuclei of the leucocytes blue, and the parasite blue.

BILHARZIA HÆMATOBIA (or distoma hæmatobium) is a trematode giving rise to recurrent hæmaturia (§ 301), inhabiting the blood of most of the Fellah and Coptic population in Egypt. The ova are extremely plentiful in the urine and are very characteristic; they measure 12 μ long by 4 μ broad, are spiked at one end (Fig. 90, p. 513), and occur in the blood and in some of the organs; the parasite itself is found in the portal system, especially in the venous plexuses about the rectum and bladder.

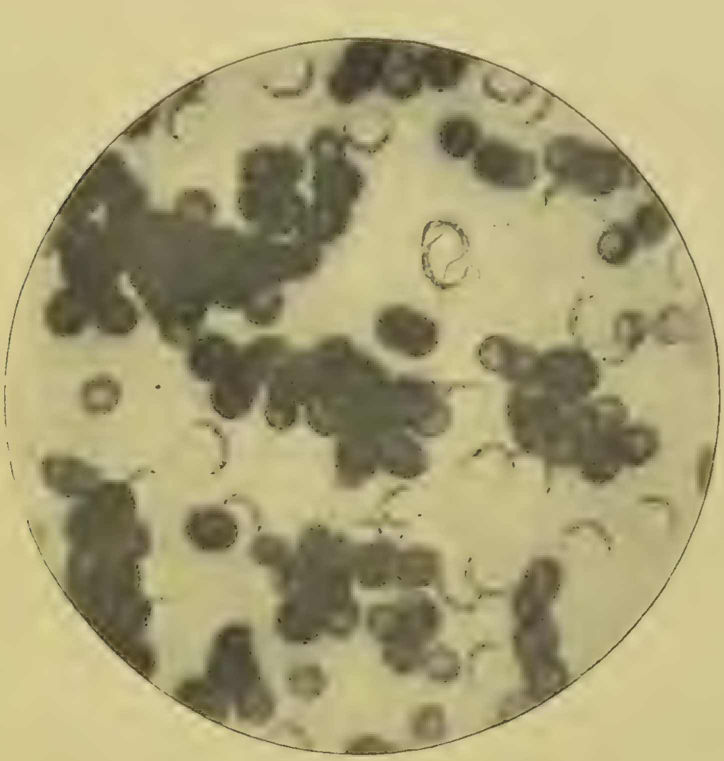

Fig. 118.—TRYPANOSOMA in blood of rat.
Specimen prepared by Dr. Emilio Eccheverria; photo-micrograph by Mr. Fred. Clark.

FILARIA SANGUINIS HOMINIS is a parasite which occasionally produces elephantiasis, chyluria, etc. The method of revealing the embryo is to allow a thick drop of blood spread upon a slide to dry. Stain half a minute in a 2 per cent. solution of methyl blue. Decolorise if necessary in dilute acetic acid (4 drops in 1 oz. of water), and examine with low power (see Fig. 117) Six species of filaria are known to infect man.

(1) The *Filaria Bancrofti* vel *Filaria Nocturna* very rarely causes any pathological symptoms in the embryonic state. The embryos may be found in 20 per cent. of apparently healthy residents in Barbadoes and other tropical climates. The embryo comes into the peripheral blood at night (from 6 p.m. to 10 a.m.); their maximum number is usually found about midnight. It may be necessary to make repeated examinations at intervals of 2 hours to be able to find them. During the day the filaria go into the lung blood vessels where they are usually found in autopsies of people who had them, and died in the day-time. Should a victim of the parasite alter his usual habits and sleep during the day the filaria periodicity is reversed. The adult filaria inhabit the

lymphatics where they give birth to immense numbers of embryos, a large number of which must in some unknown way disappear or else the blood would contain them in incalculable numbers. Embryos show a very active movement, but this is within their sheath, so that there is no locomotion properly speaking. It is this motion of their sheaths which keeps the blood corpuscles moving away from the parasite. Sometimes these embryos are actually seen escaping from their sheath, and then they become locomotive. The parasite is about $\frac{1}{80}$ of an inch in length by [illegible] of an inch in diameter. The sac prevents the creature from piercing the walls of the blood vessels, and thus gives it the chance of gaining access to the body of a mosquito, where it undergoes a metamorphosis resulting in the formation of a mouth, alimentary system and a trilobed tail. It grows until it becomes $\frac{1}{16}$ of an inch in length, re-enters man and grows to the mature stage of its existence. Adult parasites after their death cause well-marked symptoms, viz., various forms of elephantiasis, lymphscrotum, hæmato-chyluria, chylous diarrhœa and ascites, usually related to their blocking of the lymphatic circulation.

The other species are :—(2) *Filaria Perstans*, (3) *Filaria Demarquaii*, (4) *Filaria Ozzardi*, and (5) *Filaria Loa* (probably the adult form of Filaria Diurna), which are found in the connective tissue, subcutaneous or subperitoneal, and are not known to give rise to symptoms. (6) *Filaria Diurna* is of the same dimensions and anatomical characters as filaria nocturna, but it is found in the blood of patients during the day and not in the night.

TRYPANOSOMA. The parasite of *trypanosomiasis* (a disease of West Africa characterised by irregular pyrexia, erythematous rash and enlargement of the liver and spleen) and of *sleeping sickness* (a disease indigenous to tropical Africa) is a flagellated protozoon (Fig. 118). It is a minute worm-like organism found free in the blood, moving with a screw-like progress. One end of the parasite is drawn out into a whip-like process, the flagellum : the other end is bluntly conical ; the body itself is short and thick and its substance granular. Attached to one side is a transparent, flange-like process, the undulating membrane. At its posterior end is a highly refractile spot, the vacuole. The length of the parasite, including the flagellum, is about 18 μ to 25 μ. It is best stained by Leishman's or Romanowsky's stain. In sleeping sickness the organism is found chiefly in the lymphatic glands ; it causes a polyadenitis which accounts for the glandular enlargements which invariably accompany the disease.[1] When the trypanosoma gains access to the cerebrospinal fluid (although it may have previously existed for years in the blood) it is said that the patient develops sleeping sickness.

The protozoa of KALA-AZAR are found in the spleen, liver, bone-marrow, and in the lymphatic glands from the mesentery. They have been chiefly studied in blood and pulp withdrawn from the spleen, or preferably the liver during life. The commonest form found is a small ovoid body longer than it is broad, below 2 μ in diameter, measuring about $\frac{1}{3}$ of a red corpuscle in its longest axis. It contains two nuclei ; one is small, rod-shaped, and stains deeply ; the other is larger, rounded, and stains less deeply. Other forms met with consist of small groups of similar bodies clumped together resembling a quartan sporulating malarial parasite : at times these are seen breaking up into the simpler forms. A still earlier stage of this sporulisation is seen, in which pairs of unequal sized nuclei are grouped within a single cell, but with no signs of division apparent.[2] They stain faintly with methylen blue ; but the best method of staining them is by Leishman's or Romanowsky's stain. The same bodies have been found in Delhi boil and Dum-dum fever. They are known as Leishman-Donovan bodies, and are now supposed to be an early stage of a trypanosoma.

§ 401. **Physical and Chemical Properties of the Blood.** The ALKALINITY OF THE BLOOD can be tested by methods which can only be performed in a laboratory.[3] The blood is never acid, but the degree of its alkalinity varies. It is greater in men than in women and children ; it is diminished after violent exercise and the prolonged use of acids ; and it is increased at the beginning of digestion, and after the prolonged use of alkalies. In disease there is diminished alkalinity found with leukaemia, pernicious anaemia, anaemia, diabetes, cancer, great cachexia, poisoning with carbon monoxide and with acids, high fevers and various toxic processes. In chlorosis it is little if at all diminished.

The normal SPECIFIC GRAVITY of the blood is approximately 1055. It may be estimated by mixing chloroform and benzol till the specific gravity of the mixture reaches 1055, and adding to this mixture a drop of blood from a pipette. If the drop remains without rising to the surface or falling to the bottom the specific gravity of the blood is 1055. If it sinks, continue to add chloroform drop by drop, shaking the mixture the while, until the drop becomes suspended. If it floats add benzol until the drop is suspended. Then take the specific gravity of the mixture, and this is the same as that of the specimen of blood.

Significance of altered specific gravity. The amount of hæmoglobin can be estimated by finding the specific gravity of the blood, because it has been found that the specific gravity varies in proportion to the amount of hæmoglobin present. The following table[1] is of use :—

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[1] *B. M. J.*, 1902, vol. ii., p. 881 ; 1904, vol. i., p. 1252.
[2] *B. M. J.*, vol. i., 1904, p. 1249, and 1903, vol. i., p. 1252.
[3] Dr. Robert Hutchison ; see *The Lancet*, March 7, 1896.

Sp. Gr.		Hæmoglobin.
1030	=	20 per cent. ±
1035	=	30 ,, ,,
1038	=	35 ,, ,,
1041	=	40 ,, ,,
1042·5	=	45 ,, ,,
1045·5	=	50 ,, ,,
1048	=	55 ,, ,,
1049	=	60 ,, ,,
1051	=	65 ,, ,,
1052	=	70 ,, ,,
1053·5	=	75 ,, ,,
1056	=	80 ,, ,,
1057·5	=	90 ,, ,,
1059	=	100 ,, ,,

This method is of no use in cases of (i.) dropsy, (ii.) leukæmia, where the specific gravity is relatively higher than the hæmoglobin because of the weight of the leucocytes; and (iii.) pernicious anæmia, where the hæmoglobin is 20 per cent. higher than that gauged by specific gravity because of the high colour index of each corpuscle.

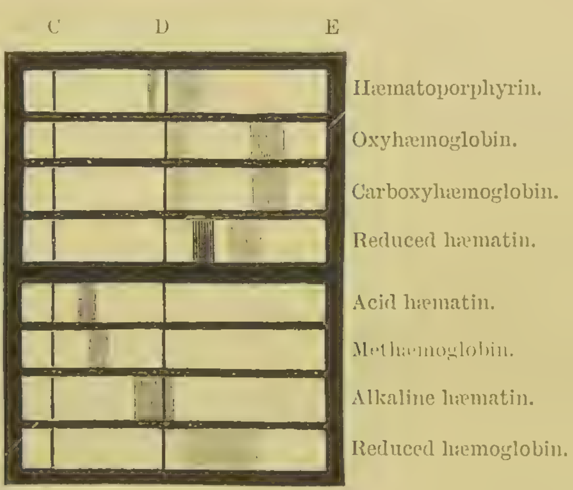

Fig. 119.—Blood spectrum (from Waller's "Human Physiology").

Spectroscopic examination of the blood. The instrument chiefly used for clinical purposes is Browning's spectroscope. It is used by holding up a glass containing a very dilute solution of blood and looking through it at the light or at a white cloud with a spectroscope placed between the blood solution and the eyes. Hæmatoporphyrin (Fig. 119) has been found in cases of sulphonal poisoning. The discovery of methæmoglobin in the blood may be a means of warning the physician of the near onset of coma in diseases such as uræmia or diabetes. Methæmoglobin is formed in amylnitrite, and potassium chlorate poisoning; and carboxyhæmoglobin in coal-gas poisoning.

The coagulability of the blood is estimated by Prof. A. E. Wright's coagulometer.[2] It has a number of fine tubes into which blood is drawn at definite intervals, and at varying times the operator blows down the tubes. When the blood cannot be blown out it has coagulated. The coagulation time is thus readily calculated. Coagulation depends upon the presence of lime salts and fibrinogen which are contained in the blood plasma, and of nucleo-proteid which is contained in the substance of the leucocytes and blood platelets. The nucleo-proteid is liberated when the leucocytes are disintegrated, and this may occur when infective toxins circulate in the blood, when the blood vessels are diseased, and in cachectic states, and thus thrombosis may in some cases be explained. Coagulation within the body is also hastened by the addition of certain salts such as calcium chloride. Coagulation within the body is diminished in certain infective diseases, and in urticaria, prurigo and allied erythematous conditions, and the administration of calcium chloride in 20 grain doses as the author has shown[1] is of great use in alleviating prurigo and urticarial conditions.

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[1] Cabot, "Clinical Examination of the Blood," 1898. Modified from Schmaltz, "Pathologie des Blutes," Leipsic, 1896.

[2] Hawksley supplies the instrument, with directions.

The FREEZING POINT OF THE BLOOD is normally —0·56° C. It is said to be lowered to —0·60° C. in cases of renal disease in which an excess of urea and salts is present, and to reach —0·8° C. in cases of impending uræmia.

CHOLINE is found in the blood in increased quantity when nerve cells degenerate (Halliburton and Mott), and may be found by laboratory methods in 10 c.c. of blood in disseminated sclerosis, myasthenia gravis and transverse myelitis, but not in hysterical affections which are apt to simulate these diseases.

Williamson's CHEMICAL TEST FOR DIABETIC BLOOD has been found useful. Make the following mixture—Blood 20 cmm., aqueous methylen blue 1 c.c., liq. potassæ (sp. gr. 1058) 40 c.c., water 40 c.c. Stand the tube containing this mixture in boiling water for four minutes. If the blood is diabetic the mixture becomes yellow; normal blood does not.

PART C. DISEASES WHICH GIVE RISE TO GENERAL DEBILITY, WITH OR WITHOUT ANÆMIA AND EMACIATION, THEIR DIAGNOSIS, PROGNOSIS AND TREATMENT.

§ 402. Routine procedure and **classification.** Here as elsewhere we have three points to investigate:—

First, the LEADING and perhaps the only SYMPTOM complained of by the patient will be debility, or pallor of the skin, or loss of flesh.

Secondly, the HISTORY OF THE ILLNESS, its date and mode of onset, and mode of evolution. Often these data are vague, but special enquiries should be directed to the condition of the digestion in times past, and any other points relating to nutrition.

Thirdly, the PHYSICAL EXAMINATION of the patient, commencing with that physiological system to which the results of our previous enquiries have directed attention, and then going through all the systems seriatim. An examination of the blood should be made in all anæmic or doubtful cases, viz., blood counts, hæmoglobin, and films.

Classification. If anæmia is suspected or PALLOR OF THE SKIN is the leading symptom, turn first to § 403.

If LOSS OF FLESH is most prominent, turn to § 416.

If GENERAL DEBILITY (without obvious pallor or loss of flesh) is most prominent, turn to § 419.

GROUP I. ANÆMIC DISORDERS.

Anæmia is an alteration in the composition of the blood, the leading character of which is a deficiency in the hæmoglobin.

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[1] *The Lancet*, August 1, 1896.

The disorders giving rise to anæmic pallor of the skin are as follows:—

COMMONER.

I. Primary anæmia (Chlorosis).
II. Pernicious anæmia.

Latent constitutional conditions.

III. Constitutional syphilis.
IV. Lead poisoning.
V. Latent tuberculosis.
VI. Latent carcinoma.

Latent visceral disorders.

VII. Gastro-intestinal conditions.
VIII. Chronic aortic disease.
IX. Chronic renal disease.
X. Chronic hepatic disease.

Recognised by history.

XI. Hæmorrhage, hyperlactation, and other causes of long-continued drain.
XII. Post febrile anæmia.
XIII. Chronic suppuration, septic processes, and lardaceous disease.

RARER (*in this country*).

With signs in spleen and lymphatic glands.

I. Leukæmia.
II. Hodgkin's disease or lymphadenoma.
III. Splenic anæmia.

With signs referable to skin.

IV. Scurvy.
V. Hæmophilia.
VI. Addison's disease and other maladies mentioned in Groups II. and III.

With typical history.

VII. Malaria and other tropical and parasitic diseases.

Anæmias of infancy.

I. Primary anæmias.
II. Secondary anæmias.
III. Congenital anæmia.
IV. Infantile scurvy.
V. Anæmia splenica infantum.

The first two disorders given in the above list are called primary, essential, or idiopathic anæmias, because they are not known to be preceded by any other disease (such as tubercle, malaria, etc.); all the others are called secondary anæmias. The division into idiopathic and secondary anæmias is somewhat artificial, and idiopathic anæmia, the anæmia of unknown causation, will probably disappear with the march of science just as idiopathic peritonitis has done. This, like all the other anæmias, is the result of a defect of the hæmogenic or the hæmolytic functions of the body about which we shall probably know more in the course of a few years.

Among the various members of the above list, the pallor may present to the experienced observer a difference in kind and degree in the several affections. Thus the greenish-yellow colour of chlorosis, the lemon yellow of pernicious anæmia, the earthy tint of carcinoma, the sallowness of aortic disease and interstitial nephritis, the pasty white of parenchymatous nephritis, and the transparent waxy look of lardaceous disease are very suggestive to the careful observer. The microscopic examination of the blood also reveals differences which are mentioned below. Among the *commoner* anæmic disorders it will be observed that after the two primary anæmias come four constitutional conditions, then four visceral disorders, and finally the three conditions which are readily recognisable by their history. The age of the patient will often give us a valuable clue.

Among the (in this country) *rarer* disorders, leukæmia, lymphadenoma, and splenic anæmia present physical signs referable to the spleen or lymphatic glands and leucocytosis: scurvy, hæmophilia, and Addison's disease present signs referable to the skin: and the remainder reveal a history of tropical disease or parasites.

The blood shows marked diminution of hæmoglobin *and other changes, and there is* NO DISCOVERABLE PRIMARY ORGANIC LESION—the disease is probably CHLOROSIS OR PERNICIOUS ANÆMIA; the age and sex of the patient and the colour-index of the blood being very different in the two diseases.

§ 403. I. **Chlorosis** ("green sickness," "poverty of blood," sometimes spoken of as primary, essential, or idiopathic anæmia) is a chronic malady in which the main feature is a diminution of the colouring matter of the blood, unaccompanied by any gross lesion, not preceded by any primary or causal disease, and occurring mostly in young women.

The chief *symptoms* are :—(1) Failing strength of vague onset and some duration, accompanied by pallor of the surface, but unaccompanied by wasting. There is indeed often an excess of adipose tissue. The pallor is marked in the lips, gums and conjunctivæ (as may be observed by pulling down the lower lid), and the sclerotics have a bluish colour. The skin sometimes presents a greenish hue, hence the name chlorosis (χλορος, green). These patients generally present themselves on account of either cardiac or digestive symptoms, headache, or amenorrhœa. (2) Cardio-vascular symptoms, such as dyspnœa on slight exertion, palpitation, and a tendency to syncope. In a fair proportion of the cases dilatation of the heart can be made out. Hæmic murmurs are heard, especially over the pulmonary area. In marked cases the "bruit de diable" is to be heard—a continuous hum heard when the stethoscope is gently placed over the jugular vein in the neck. Œdema of the ankles at night is common, and venous thrombosis may be met with.

Hæmic or anæmic murmurs may be either systolic or double in rhythm, never diastolic alone or presystolic. They are usually soft and blowing, but may be extremely loud and rasping; loudest in the pulmonary area, but they may be heard all over the precordium, very rarely in the axilla; often louder when the patient is lying down or has rested, and apt to vary from day to day. As regards their causation, my own belief tallies with those who hold that they are due partly to increased fluidity of blood, and partly to a flabbiness or enfeeblement of the cardiac wall. The wall, therefore, yields to the intra-ventricular pressure, and the resulting dilatation of the orifices leads to regurgitation. Dr. Drummond (*Lancet*, 1895, ii., p. 193) recognises three classes of functional murmurs: 1, cardio-hæmic, or anæmic; 2, neuro-typtic, or cardio-muscular, or shock-bruit—the result of excited muscular action; 3, cardio-respiratory.

(3) Disturbances of digestion are generally present, such as deficient or capricious appetite, and discomfort after food; and there may be atonic dyspepsia, gastric atony and gastroptosis. Prolonged and intractable constipation almost invariably precedes and accompanies the affection, and is said by some to be its principal cause. (4) Symptoms referable to the nervous system, such as headache, neuralgia, vertigo, defective attention, nervousness, irritability or depression of spirits, spots before the eyes. (5) Amenorrhœa is usual, dysmenorrhœa not infrequent; menorrhagia sometimes precedes and accompanies chlorosis. (6) Some authorities say that chlorosis may be attended by elevation of temperature, from time to time, but adequate proof in the post-mortem room should be forthcoming that such cases have not been due to some undiscovered syphilitic, tuberculous, or septic lesion (*vide* p. 643, footnote). In ordinary cases the temperature is subnormal. (7) *Blood changes.* There is usually but little diminution in the number of red corpuscles, though a great diminution in the hæmoglobin which they contain. The red corpuscles appear pale, and the total hæmoglobin in the blood is sometimes as low as 30 per cent. of the normal. In severe cases, however, there is also a considerable diminution (down to 2,000,000) in the number of red corpuscles. Poikilocytosis, leucocytosis and nucleated red cells are uncommon except in severe cases (see § 399); chemical analysis shows a marked diminution of the iron in the blood.

The *diagnosis* is not as a rule difficult, by reason of the age and sex of the patient. The pallor of the skin, though it may have a yellowish green tint, is usually quite different from that of jaundice, or the cachexia of malignant disease. It has at times to be differentiated from that due to any of the conditions mentioned in the succeeding sections below, and especially from gastric ulcer. The chief danger is lest an early stage of chronic tuberculosis should be overlooked; and to avoid this a thorough examination of all the organs should be made; the principal physical signs in chlorosis being the murmurs and the pallor. The pallor of renal disease is an ivory white, and is attended by albuminuria, tube-casts, and in some forms by generalised dropsy. Chlorotic girls may have œdema of the ankles, but there is no albumin in the urine. The diagnosis from organic heart disease is given under cardiac murmurs (vol. i., p. 92).

Prognosis. The disease is rarely fatal, but it is extremely liable to relapse, and the symptoms are sometimes so grave as to necessitate strict confinement to bed. It is essentially a chronic condition, and its course always extends over many months, unless checked by treatment. Gastric ulcer is a frequent sequel. Thrombosis of the femoral vein may occur, and, occasionally, of the longitudinal sinus. "Spurious haemoptysis," *i.e.*, haemoptysis without any pulmonary mischief, probably coming from the mouth or throat, occurs occasionally.

Etiology. The disease is practically confined to young women between the ages of 15 and 25, who have suffered from chronic constipation, and have not sufficient out-of-door exercise, or fresh air. It is a curious and unexplained circumstance that more blondes than brunettes are affected. The disease has at different times been thought to be due to a small aorta, the onset of puberty, chronic constipation, or the wearing of tight corsets. It has been suggested that in constipation the iron in the food does not reach the system, owing to its decomposition in the alimentary canal, and its combination with sulphur to form sulphides. The marked benefit derived from respiratory exercises, suggests deficient oxygenation.

The *treatment* of chlorosis consists, first, in correcting any digestive trouble which may be present, or constipation, or intestinal sepsis (*e.g.*, by naphthol or salol); secondly, in the administration of iron; and thirdly, in adopting hygienic measures, especially such as promote oxygenation. It is well to remember that at least 3 months are required to effect a cure, and the patient should be warned of the danger of relapse. Iron should be administered in sufficient quantities and in gradually increasing doses (*e.g.*, one Blaud's pill, F. 86, thrice daily for the first week, two for the second, three for the third, and so on for five weeks, then decreasing the dose). Liquor ferri perchlor. is a very valuable remedy, if the stomach will tolerate it, on account, it is said, of the amount of free chlorine given off which acts as an intestinal antiseptic. It may be combined with mag. sulph. Ferri sulph. (alone or with aloes), one grain thrice daily for the first week increased to two grains for the second and three for the third, continuing at nine grains per diem for three months, will seldom fail to effect a cure. If nervous symptoms be present, syr. ferri phosph. is useful; while ammonio-citrate or peptonate of iron or reduced iron may be given to patients with delicate stomachs. When menorrhagia is present, treatment directed to this condition should be adopted (p. 554). If the case resists iron in one or another form, the diagnosis should be revised, and tubercle or one of the other conditions mentioned below considered. Other remedies include arsenic, cod-liver oil, lacto-phosphate, malt extract, manganese preparations, dilute acids and the administration of oxygen. Hypodermic injection of iron is much in vogue on the continent, and good results are reported therefrom. Rest in bed is necessary in severe cases; and complete rest alone in many cases is of great benefit, for the calibre of the arteries is enlarged thereby and dilatation of the heart is prevented.[1] The curative effects of fresh air must in nowise be forgotten. The patient should sleep with window open, and be always in the open air. For mild cases gentle exercise for both mind and body should be prescribed. Late hours, tight lacing, overstudy, worry and idleness are to be forbidden. To enlarge the capacity of the chest give respiratory exercises. The diet must be liberal, especially in regard to nitrogenous food in the form of butcher's meat (which should be taken at least once daily), and milk. Green vegetables and fruit are useful. The patient should avoid drinking much at meal times; half a tumbler of fluid is sufficient. Two to three hours after food a tumbler of hot water may be taken. The food should be carefully masticated, and the teeth attended to if necessary.

§ **404.** II. **Pernicious Anæmia** (syn. Idiopathic anæmia of Addison, Addison's anæmia) is a relatively rare variety of primary anæmia first described by Addison (who called it idiopathic anæmia) and now known to be associated with certain other signs of toxæmia and of hæmolysis, chiefly affecting men in the second half of life, and running a chronic and generally fatal course. The three principal changes constantly present after death are—(1) the heart exhibits fatty degeneration; (2) the bone marrow is unduly red and contains a great number of nucleated red cells, especially gigantoblasts; and (3) the liver is fatty, sometimes enlarged, and contains an excess of iron deposited within it, as shown by the ferrocyanide reaction. Extensive atrophy of the gastric mucosa may also be found. Changes have been discovered in the sympathetic ganglia, and in the posterior columns of the cord by Lichtheim and by Burr.

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[1] See Dr. G. Oliver's book on Pulse Gauging, p. 135.

The *symptoms* may be classed in several groups, viz.: 1. General weakness and anæmia of insidious onset, with their usual effects, *e.g.*, palpitation, dyspnœa, a tendency to syncope, hæmic murmurs, and other symptoms as in chlorosis (*q.v.*). There is little if any wasting; there may indeed be much subcutaneous fat. 2. General toxic symptoms, such as lassitude, irregular pyrexia from time to time (though the temperature at other times may be normal or sub-normal), tingling of the extremities, and other vaso-motor systems. 3. Oral sepsis and gastro-intestinal symptoms, *e.g.*, sore tongue, dental necrosis, and suppuration of the gums are often associated; gastric pains, and diarrhœa. The spleen is not enlarged. 4. Symptoms due to hæmolysis, *e.g.*, urobilinuria, excess of uric acid in the urine, and the lemon tint of the surface of the body. 5. There is a marked tendency to hæmorrhage from the mucous membranes, into the internal organs, the retina, and sometimes into the skin. 6. The *blood changes* are very characteristic (§ 399 and Plate IV.). (a) The red corpuscles are much reduced in number, sometimes falling to less than one-fifth of the normal. (b) The colour index is high, *i.e.*, the percentage of hæmoglobin in each corpuscle is increased, but the total hæmoglobin in the blood is diminished, *e.g.*, to 15 to 37 per cent. of the normal. (c) The most characteristic alteration in the blood consists of irregularity in size (megalocytes and microcytes) and shape of the red corpuscles (poikilocytosis); nucleated red corpuscles (megaloblasts and normoblasts) are present, with granular degeneration of the protoplasm. It has been suggested[1] that the changes may be due to the arrest of development of the red cells from the erythroblasts (nucleated red marrow cells). (d) The pale watery blood of chlorosis coagulates rapidly, but that of pernicious anæmia very slowly, and in the latter the corpuscles show no rouleaux formation. (e) Leucocytosis is not present except in the very severe forms.

The *diagnosis* of pernicious anæmia is effected by the age and sex of the patient, and by the characteristic blood changes, viz., a marked alteration and diminution in the red corpuscles without corresponding diminution in the hæmoglobin value of the blood—the converse of chlorosis. The diagnosis from some of the diseases given below, and particularly from latent cancer of the pylorus, is often extremely difficult, and can only be accomplished by an exhaustive review of the case.

Prognosis. The disease is slow but progressive, and almost invariably fatal. The patient often rallies, and hopes of recovery are entertained, but he almost always relapses. Progress may be estimated by examining the blood from time to time. A preponderance of megalocytes is an untoward sign. The main complications are visceral hæmorrhages, cerebral or spinal hæmorrhage, and degeneration in the spinal cord, chiefly affecting the posterior columns. The nervous system towards the end is considerably disturbed. Headache, nervousness, and prostration are fairly constant, but the intellect is usually clear to the end. Sometimes convulsions and coma may occur. A form of paraplegia may supervene, due to a special kind of sclerosis of the spinal cord, as Dr. James Taylor has shown.

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[1] *E.g.*, Dr. Sydney Martin, *The Lancet*, 1900, vol. i., p. 1006.

Etiology. Pernicious anæmia chiefly attacks males from 25 to 45, occasionally women of the same age; it is very rare in the young. It comes on insidiously, without apparent cause. It has been ascribed to (a) defective hæmogenesis, *i.e.*, blood formation (Stephen Mackenzie, Rindfleisch, etc.), or to increased hæmolysis, *i.e.*, blood destruction (Stockman and Hunter). The latter theory is strongly upheld by Hunter,[1] who believes the disease is due to a gastro-intestinal sepsis resulting usually from some morbid condition of the mouth, such as dental caries, or suppurative gingivitis. This, he says, leads to absorption of toxins, which, on reaching the blood destroy the red corpuscles (hæmolysis), thus giving rise to deposit of iron in the liver, urobilinuria, anæmia and other symptoms.

Treatment. Complete rest in bed is necessary. Antiseptic mouth-washes should be used, and carious teeth extracted or stopped. The diet should be nutritious and contain plenty of nitrogenous constituents, such as raw meat juice. Milk and farinaceous foods are also beneficial. Arsenic has a greater control over the disease than any other drug, but it has often been noted to be of more use in the primary attack than during a relapse; the dose should be gradually increased until a drachm of Fowler's solution is being given daily. Naphthol, salol and other intestinal antiseptics have been used with advantage. Red marrow, transfusion of blood, oxygen inhalations and subcutaneous injections of antistreptococcic serum [2] also have been tried.

The patient is PALE, *but the anæmia does not quite conform to the preceding types and is* **not readily amenable to treatment by iron**—the disease is probably some LATENT CONSTITUTIONAL CONDITION (syphilis, plumbism, tubercle or carcinoma), or some LATENT VISCERAL DISEASE.

§ 405. III. **Syphilis** (syn. Hunterian or constitutional syphilis) is a constitutional malady, due to a microbic infection, which starts as a superficial ulcer (chancre) at the seat of inoculation, runs a prolonged and indefinite course, is liable to break out anew during the whole lifetime of the patient without fresh infection, even after many years of quiescence, and in its later stages produces granulomatous deposits in various parts of the body, particularly in the skin and nervous system.

Symptoms. Syphilis leads to a degree of pallor which may simulate chlorosis or other forms of anæmia very closely. When no history of primary syphilis is obtainable, and no physical signs can be discovered, the diagnosis from other forms of anæmia may be difficult. For the sake of convenience the symptoms of syphilis are divided into three stages, but it must not be forgotten that their mode of appearance is extremely variable, and that the three stages may even appear simultaneously in certain patients. *Primary stage.* The latent period—period of incubation—generally lasts about *3 weeks* but it may vary from 10 to 46 days. The initial manifestation appears as a superficial ulcer (the hard or Hunterian chancre) at the site of inoculation; it is usually single, and occurs most commonly on the prepuce or glans penis in the male, and the labiæ and nymphæ in the female. It originates as a flat, elevated, painless papule, which slowly enlarges and may desquamate without breaking down; or superficial erosion or ulceration takes place with a slight serous discharge, thus differing from the deep excavated ulcer of the "soft sore" or non-Hunterian chancre. The underlying induration of the tissues is always a marked feature, hence its name "hard sore." The lesion after a time cicatrises and usually leaves behind it some slight discoloration or induration, or both, which mark the site. Sometimes the primary sore is so slight as to be overlooked and appears to be wanting, especially in the female. About the same time or within 1 or 2 weeks the associated lymphatic glands, usually in the groin, become enlarged and hardened. Even thus early in the disease, the red blood discs are generally diminished to 3 million per cub. mm. or even less, and there is pallor and weakness, conditions which increase as the disease progresses if untreated. The glandular enlargement may become generalised, and may persist for months or years, and this hardness of the lymphatic glands may thus serve as an aid to diagnosis at any time.

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[1] *The Lancet*, 1901, vol. i., pp. 473, 930, and 1903, vol. i., p. 283.
[2] See *The Lancet*, 1901, vol. i., p. 930.

The *secondary* symptoms make their appearance about 3 weeks after the first appearance of the chancre (4 to 12 weeks after inoculation). In typical cases a faint generalised dusky macular rash (which may be brought out more distinctly by a warm bath) appears chiefly on the chest and abdomen, nearly always attended by sore throat, and often attended by malaise, pains in the limbs, anæmia, and slight pyrexia. The rash takes about 3 weeks to mature and 3 weeks to decline. The pyrexia, which is generally overlooked, has already been described (§ 383). The sore throat is usually of an indolent, ill-marked kind, with whitish secretion resembling snail tracks on the tonsils (§ 116). The eruptions which may appear now and hereafter are of many different kinds—

macular, papular, scaly, pustular, tubercular, practically never eczematous or vesicular. The characteristics of these (see also Chapter XVIII.) are their reddish brown colour, generalised or symmetrical distribution, grouping in segments of circles, and their preference for the forehead and flexor surfaces, their polymorphism and absence of itching. The hair may fall out and the nail beds be affected with an indolent inflammation. Moist "mucous patches," with a highly contagious secretion, are apt to appear at the corners of the mouth and other mucous orifices. The diagnosis of the skin symptoms (§ 488) and the lesions of the mucous membranes (§ 149) are dealt with elsewhere. The eyes may become affected by iritis, choroido-retinitis, and the bones with periostitis in which the pain is worst at night, the nervous system and the viscera with gummata, and the joints with synovitis. Any of these symptoms may crop up again and again during the ensuing months or years.

Later Stages (so-called tertiary symptoms) and *varieties* of syphilis. In practice it is convenient to recognise two broad varieties of syphilis. In most cases of a BENIGN type, *adequately treated* there is no recurrence of symptoms after the second stage above described; in short there is no tertiary stage. But in other cases the disease assumes a MALIGNANT type either by reason of the intensity of the virus or the predisposition or debilitated state of the individual, combined perhaps with inadequate treatment in the earlier stages, and such cases are characterised by severity of the initial symptoms and a tendency to recurrence at intervals throughout life. It is in such cases more especially that one meets with what are known as "tertiary" symptoms. Malignant or tertiary lesions, as exemplified for instance in the skin, are characterised by having a greater and deeper infiltration, a greater proneness to suppuration, ulceration, and scarring, and by being followed by more loss of tissue than the benign lesions. All the same skin symptoms noted in the secondary stage may recur, but they are more apt to be localised and asymmetrical in distribution, serpiginous in outline, lenticular or nodular in shape, and pustular or ulcerating in character than the corresponding secondary symptoms (Chapter XVIII.). Nodular or infiltrating gummatous deposits followed by scarring and perhaps by ulceration may affect the mucous membranes, particularly in the oral cavity and its diverticula, the liver and other abdominal organs, and the cephalic and the genito-urinary organs, and lead to fibroid degeneration, stricture, or destruction of the proper tissues and functions of the parts. The bones are often attacked by gummatous periosteal deposits, leading in the case of the hard palate to perforation, and in the other flat and the long bones to the formation of "nodes." The arteries may become thickened and arterioles blocked; partly for this reason, partly by the proneness of the virus for the nerve-tissues and meninges, the nervous system is specially apt to be involved. But even this list does not complete the account of this insidious, prolonged and terribly far-reaching disease, for it is, as mentioned in previous chapters, one of the two causes of lardaceous disease of the liver, spleen, kidneys, and intestines. More or less anæmia is a symptom throughout the disease, and in untreated or malignant cases of syphilis the cachexia may sometimes be fatal, as in patients referred to on p. 735 and elsewhere.

Para-syphilitic (παρα = derived from) is a term applied to certain diseases which are not due to definite syphilitic deposits or infiltrations, but are nevertheless due to the indirect effects of the syphilitic poison on the system, or to its after effects. Thus locomotor ataxy is due to the after effects of the virus on the muscle sense neurons. Paralytic dementia and certain types of epilepsy are also examples of para-syphilitic disorders.

Hereditary or congenital syphilis (compare Table I. and facies of disease, pp. 26 and 32). We have seen how ubiquitous the consequences of syphilis may be, but it is upon the children of such parents that the heaviest nemesis falls. Happily, the mother very often aborts, syphilis being one of the commonest causes of abortion, or the child dies and decomposes within the uterus, or being born alive it dies in the first 12 or 18 months of life of marasmus or its complications. Thus a series of miscarriages or still-births, or a heavy mortality among children in the early months of life, imply a strong probability of syphilis in the parent. If the child be born alive the primary chancre is of course wanting, but the symptoms conform more or less to the secondary symptoms above described. The infant is sometimes

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healthy at birth but in a few weeks it develops "snuffles," or a ham-coloured eruption on the buttocks, flexures, palms, or soles, and marasmus sets in and is followed (if active treatment is not adopted) by any of the other secondary symptoms above mentioned. The child is fretful, the cry is hoarse, and the bones are tender; and gastro-enteritis, bronchitis or pneumonia may complicate matters. If the child survives the first 12 or 18 months of life, a long period without fresh syphilitic manifestations ensues, excepting perhaps in the rapid decay of the temporary teeth and stunted growth of body. About the seventh year however, the permanent teeth appear and usually present the pegged shape and notched border described by Mr. Jonathan Hutchinson, Senr. (Fig. 3, p. 25). Again, there may be an interval of quiescence, but about the 14th year or puberty, interstitial keratitis, deafness, periostitis or synovitis may appear; the skin, viscera, and nervous system only rarely being affected. From this time onwards the evidences of hereditary syphilis consist of the consequences of the previous lesions on the general development, the skin, the mucous orifices, the malformation of the bones, the eyes, ears, and teeth, which are summarised in Table I., p. 34.

The *diagnosis* of the Hunterian chancre will be found in surgical works. The diagnosis of syphilitic symptoms and lesions in the skin, nervous system, liver, and other parts, will be found in the appropriate chapters of this work. The existence of hard shotty glands as an aid to diagnosis has already been referred to. In regard to the diagnosis of syphilitic from other forms of anæmia undoubtedly mistakes may easily be made. I remember the case of a very anæmic lad of 15 in whom the only other symptom besides those of anæmia was a slight rise of temperature in the evening, which was diagnosed as one of pernicious anæmia; after death, however, gummatous deposits were found in the meninges in the frontal region and elsewhere. A somewhat similar case of gumma of the liver was reported by the late Dr. J. S. Bristowe.[1] Syphilis should always be suspected in obscure cases of anæmia, and very often the amenability to treatment by iodide and mercury will be a revelation. The presence or history of an eruption should be noted and the viscera, the bones and the eyes very carefully examined.

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[1] Clinical Soc. Trans., vol. xix., p. 249.

To identify the syphilitic origin of anæmia in cases presenting no other sign of the disease, Justus proposed a test which at one time seemed likely to be of service.[1] He found in constitutional syphilis, as soon as the virus had resulted in glandular enlargement and when the patient had not recently been treated by mercury, that intravenous or intramuscular injections of mercury led to a breaking down of the red cells and consequently a lowering of the amount of hæmoglobin in the blood. The hæmoglobin of the suspected case is first estimated (§ 397) and an injection of mercury administered, then the hæmoglobin is again estimated 3 times at intervals of 12 hours, and in syphilis there is a reduction of 10 to 20 per cent. Unfortunately this mercury-hæmoglobin reaction has not been found in all cases of syphilis and it has also been revealed in some non-syphilitic anæmias, Dr. Cabot (*loc. cit.*) having found it in a case of chlorosis, and Drs. Tucker and Huger[2] in a case of herpes and one of non-syphilitic genito-urinary disease.

Prognosis. Syphilis is never fatal (excepting, perhaps, in the case of the fœtus) by the intensity of its toxæmia, like small-pox or scarlatina; but it may kill in infancy by marasmus. In adults it only kills, usually after a life of invalidism, by its complications or by involving some vital part. Benign cases of the disease adequately treated, if the patient lives a temperate, hygienic life, may give no after trouble, and many such persons live to old age and have perfectly healthy children. Nevertheless, it behoves even these patients to be constantly on their guard, for once syphilitic means that they are always liable to the possibility of recurrence, even to the end of their days. Malignant types of the disease are sure to recur, and to require active treatment on and off throughout life. The severity and duration of an attack of syphilis are influenced by a number of circumstances, some of which are hard to gauge. The habits and mode of life (especially as regards intemperance), age, occupation, exposure, privation, pre-existing disease (especially tuberculosis and renal disease), all doubtless influence the course of the malady. The disease is often said to prevail in a particularly virulent form in some naval and military stations. But of all factors, the one which influences the prognosis of syphilis more than anything else is adequate and continuous treatment during the earlier phases of the malady.

Etiology. The specific microbe has not yet been satisfactorily identified, but syphilis resembles the specific fevers in having a period of incubation, followed by a fairly characteristic eruption, and in the fact that one attack renders a person immune to a second attack, with very rare exceptions. It differs from other specific fevers in the extreme length of its course, which may last many years, in the long intervals which may separate its various manifestations, and above all in its curious liability to recur without fresh infection. Inoculation can only take place through an abrasion of the skin or mucous membrane, and may occur in three ways: (a) usually it is by direct contact with an infected person, generally during sexual intercourse, but in some cases (*e.g.*, in doctors and midwives) as the result of examining diseased persons, by suckling (as in wet nurses), kissing, etc.; (b) occasionally by the use of contaminated articles, *e.g.*, spoons, cups, pipes, towels, surgical instruments, or (?) the seat of a w.c. In the first two stages the blood and the *moist exudations of all the lesions are certainly contagious.* In the later stages, some difference of opinion exists as to the contagiousness of the blood and secretions. My own experience is that all moist lesions in all stages (and therefore the blood also) are contagious, and I well remember a patient of mine who had contracted the disease 33 years before, and who conveyed the disease to his sister by kissing, he having at the time only a small fissure at the angle of his mouth. In regard to marriage, I see no reason to modify the rule about to be stated, however old-standing the disease may be. The use of human vaccine lymph, even when free from blood, for vaccination purposes from arm to arm has undoubtedly been the occasional means of propagating syphilis, but the frequency of the occurrence has as certainly been exaggerated. (c) To the offspring syphilis may be conveyed by hereditary transmission of the virus from either parent. If, as frequently happens, the mother becomes infected shortly before she becomes pregnant, or during the early months of pregnancy, the child seldom escapes the disease; if after the seventh month, however, the child may be healthy. It is said that the child may be infected from the father, and that the mother may nevertheless escape; but this is extremely doubtful, for under these circumstances the mother never contracts the disease from her child after its birth (Colles' law). The date when marriage is permissible is a most important one. It should under no circumstances be sanctioned within 2 full years after infection, even in the mildest case. Subsequently, if the patient has been free from any syphilitic symptoms for at least 12 months (Jonathan Hutchinson), and has been undergoing anti-syphilitic treatment during that time, there is every prospect that the offspring will be healthy. It is a good thing to insist on this interval of quiescence as a minimum, combined with this period of treatment, before sanctioning marriage or possible conception.

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[1] Quoted by Otto Grünbaum, *West London Med. Journ.*, April, p. 86, and discussion, p. 109, and by Cabot, *loc. cit.*

[2] "Medical Annual," 1904, p. 671.

Syphilis has, there is little doubt, prevailed from very ancient times, and has occasionally occurred in the form of widespread and severe epidemics, particularly when introduced into previously healthy communities. Individuals of all races and ages are subject to it, but the disease cannot be transmitted to the lower animals. Extremes of temperature seem to intensify its virulence. Tuberculous and otherwise debilitated subjects usually incur syphilis in a severe and malignant type.

Treatment. Fortunately, in mercury and iodine we have efficient remedies for controlling this serious and far reaching disease. The most effectual remedy in the *earlier stages* is mercury, which should be given continuously for at least 18 months, whether symptoms be present or not (Mr. Hutchinson gives 18 months, Dr. W. Osler 2 years), and subsequently, if symptoms are still present, until at least 3 months after all evidences of the disease have ceased. It may be given by mouth, by inunction or fumigation, and by hypodermic injections into the muscles or veins. The precautions to be observed in all cases, in addition to the duration of the treatment just mentioned, are: (i.) The mercury must be gradually increased until the gums become tender, then regulated until toleration is established. It is well to get rid of decayed teeth at the very outset, and a mouth-wash of pot. chlor. and weak carbolic acid may be necessary. (ii.) The patient should be seen once or twice a week so as to watch for salivation, diarrhœa, gastric disturbances, and to regulate the treatment. If the mercury be temporarily stopped, 5 grains of iodide of potassium should be given thrice daily. Debilitated subjects and subjects of renal or visceral disease require smaller doses and extra caution. (iii.) The patient should absolutely avoid alcohol and tobacco while under treatment, and take plenty of milk and light food. The open-air treatment as for tuberculosis is given at Aix-la-Chapelle. Sea air, and especially sea voyages, are specially beneficial.

Mercury is ordinarily administered by the mouth, liq. hydrarg. perchlor. (with or without potass. iod.) being given thrice daily after meals, gradually increased; or hyd. c̄. cret. gr. j. or ij., or pil. hydrarg. gr. j. or ij. may be given with opium gr. $\frac{1}{10}$ twice daily. In private practice a convenient way to give mercury is by means of a pill of hydrarg. iodidi viridi gr. $\frac{1}{2}$ to 1, with opii gr. $\frac{1}{4}$ to $\frac{1}{3}$ twice daily. Sarsaparilla and guaiacum when added to medicines containing mercury or iodide appear to increase their effects in some cases. The inunction of ung. hydrarg. is another method; a piece the size of a Barcelona nut being well rubbed in to one or other axilla or groin every night. It is a dirty method, and does not admit of exact dosage. In infancy the favourite methods are the administration of hyd. c̄. cret. gr. j. or ij. once or twice daily, or the wearing of a broad flannel bandage on which ung. hydrarg. is spread daily. For adults a cleaner and prompter method than inunction is the mercurial vapour bath (F. 4), given daily at first.

The hypodermic introduction of mercury—which is clean, convenient and does not upset the stomach—dates from the time of John Hunter and has come into vogue again during the last few years, many different preparations having been used. I have been well content with a solution of perchloride of mercury (F. 24), to which 1 or 2 per cent. of cocaine may be added to prevent pain. An ordinary hypodermic syringe is used, though the needle is soon spoiled. The skin and the hypodermic needle having been rendered aseptic, the solution is injected into the substance of a muscle, preferably the muscle of the buttock. Start with a small dose, about one-tenth to one-fifth of a grain daily, or every other day, until the gums become affected; gradually increase the dose, and then administer about $\frac{1}{4}$ grain once or twice a week or every second week, according to the state of the gums. Some prefer the succinimide (F. 24), peptonate, cyanate,[1] cyanide,[2] or other soluble salt of mercury, and some recommend a solution of biniodide in oil.[3] The insoluble preparations—calomel, metallic mercury or salicylate of mercury suspended or emulsified in paraffin and injected by means of special syringe—have been largely used in France and

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[1] Max Stern, *Munich Med. Woch.*, July 2, 1901.

[2] Bonzitat, *Thèse de Paris*, July, 1902.

[3] Melis, "Archives Belges," 1901, p. 21, and *Brit. Med. Journ.*, 1901, vol. ii., "Epitome," p. 20.

Germany,[1] but I have found the soluble salts to be more convenient, more rapid in their action, and more exact in their dosage. L'huile grise (mercury suspended in oil) is said to be sometimes cumulative in its effects. The intravenous injection of mercurial preparations is advocated by some, but is not free from the danger of thrombosis.[2] Syphilis has been treated by the injection of serum derived by a cantharides plaster from patients with tertiary symptoms: 10 to 40 c.cm. were injected every third or fourth day for 1 or 2 months. Refractory cases are said to have improved, and the improvement to have been more lasting than after a course of mercury or iodides.

It is in the later *secondary* and in the *tertiary stages* of syphilis and particularly in the internal manifestations that potassium iodide finds its chief use, and in these, in a large proportion of cases, this alone will promote absorption if given in large enough doses. My belief is that the doses generally given are not large enough because I have known many cases which fail with 5 or 10 grains succeed with larger doses. My practice is to start with 20 grains and rapidly increase the dose to 60 grains thrice daily after meals followed by a glass of milk. If the patient has running from the nose and eyes it may often be relieved by doubling the dose. The largest dose I have given was 90 grains thrice daily. Iodide spots may sometimes be relieved by three drops of liq. arsenicalis, and as the iodide lowers the arterial tension spirit. ammon. aromat. or other stimulants may be called for. If iodide of potassium disagrees even when followed by milk, ring the changes with the sodium and ammonium salts. After the symptoms have disappeared the doses may be lowered, but on no account should the iodide be stopped until at least 3 months later. Mercury may with advantage be added in bad cases or if an insufficiency of the metal has been taken.

§ **406.** IV. **Plumbism** (syns. Saturnism, chronic lead poisoning) is a condition of anæmic pallor usually associated with a number of other symptoms, resulting from the slow absorption of lead into the system due to the contamination of drinking water or to the occupation of the patient.

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[1] Roux, *Jour. de Med. and Chir. Prat.*, May 10, 1902; Dr. Leredde, *Medical Press*, Oct. 29, 1902; Max Stern, *Munich Med. Woch.*, July 2, 1901; see *Brit. Med. Journ.*, May 30, 1903, p. 1258.

[2] Mendel, "Medical Annual," 1904, p. 22.

[3] Moore, *Dermatol. Zeitsch.*, April, 1901, and *Brit. Med. Journ.*, 1901, vol. ii., "Epitome," p. 67.

Symptoms. (1) The anæmia is very marked; the red corpuscles may be reduced to 50 per cent. The pale, pasty appearance of house-painters is well known. The anæmia of lead as in the 3 preceding causes is not very amenable to iron. (2) The gums, with but very few exceptions, show the so-called "blue line," a peculiar livid line on the margin of the gums immediately adjoining the teeth. It is due to the formation of lead sulphide. A few medicinal doses of lead, copper and other metals may produce it. Dr. Thomas Oliver[1] says the true blue line is incapable of removal by medicinal treatment under from 8 to 12 weeks. (3) Very obstinate constipation is usual in the subjects of chronic lead poisoning, and is sooner or later associated with (4) severe intestinal colic (§ 173). This colic is very apt to recur, and its recurrence is an aid to its diagnosis. (5) Lead is usually found in the urine, and chronic plumbism is frequently associated with slight albuminuria. Ultimately chronic Bright's disease supervenes. The arterial tension is high and arterial sclerosis supervenes relatively early in life. (6) Lead has a special tendency to attack the peripheral motor nerves. It has a special proclivity for the musculo-spiral nerve, and thus the typical lead palsy is a "wrist drop" due to paralysis of the extensor muscles of the forearm. It is important to note that the supinator longus generally escapes and therefore supination is preserved. Both limbs are invariably affected, though one side may be worse than the other. The muscles rapidly waste and present a typical reaction of degeneration. Other recognised types of paralysis are the brachial (or scapulo-humeral), the Aran-Duchenne (resembling chronic anterior polio-myelitis), the peroneal, and the adductor laryngeal. Sometimes all the limbs are affected so that a complete generalised paralysis, including perhaps the diaphragm, may supervene. Muscular tremor is not very common in the lead poisoning due to drinking water, but is met with sometimes in the pre-paralytic stage; it is, however, very usual when the lead is introduced into the system by inhalation, *e.g.*, among lead miners and glass blowers.

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[1] "Dangerous Trades," London, 1902.

Optic neuritis, neuro-retinitis, amaurosis, and amblyopia are met with. Cerebral symptoms (lead encephalitis) are met with, such as acute mania, convulsions, coma, and occasionally insanity.

The *diagnosis* of lead poisoning is not difficult when once our suspicions are aroused. Each of the above symptoms is fairly characteristic, and when all are met with together there can be no doubt. In doubtful cases of anæmic pallor the urine should be carefully examined for lead. The "blue line" is very characteristic.

Prognosis. The prospect of recovery varies with the duration of the disease, the severity of the symptoms, their amenability to treatment, and the condition of the kidneys. Slight palsies may be completely restored, but when there is extreme muscular atrophy and persistent absence of faradic reaction recovery is rare.

Etiology. (1) Numerous occupations afford opportunities for the introduction of the poison either into the alimentary canal or by inhalation into the lungs, such as painters, lead workers, plumbers, pewterers, glazed card makers, pottery glaziers, tile cutters, chromic dye workers, enamellers, glass workers, lead foil makers, shoe finishers, bleachers of Brussels lace, printers' compositors, and lead miners. (2) The disease may occur in epidemics owing to the consumption of contaminated water which has been stored in leaden cisterns, or has passed through leaden pipes. A specially soft water is apt to dissolve a certain amount of the lead carbonate usually found inside leaden pipes. In other epidemics, such as that of Newcastle in 1900, the water has been found to be slightly acid near its source. (3) Various articles of food or drink are apt to become contaminated when stored in lead, lead-glazed, or pewter vessels, such as tinned provisions, beer, cider, or wines. Cases still occur in men who have drunk the beer first drawn off in the morning which has lain some hours in a pewter or leaden pipe. (4) Rarer cases are the sleeping in newly painted rooms, and occasionally in susceptible persons the external application of a lead lotion may produce poisoning. The internal administration of lead as an abortifacient is said to be common in some Midland towns (Ransom). Any age or sex may be affected, but women appear to be more susceptible than men. A first attack may not occur until after exposure for many years. Alcohol predisposes.

Treatment. The first indication is the avoidance of the cause; and those who are exposed to the poison by reason of their occupation should observe the greatest personal cleanliness. The face, hands and teeth should be cleansed before meals. The ventilation of the work-room should be supervised, and a respirator worn if the air contain much dust. Dr. Oliver has a poor opinion of the prophylactic value of sulphuric acid lemonade, inasmuch as the sulphate of lead is hardly less soluble than the carbonate. Fruits and alcohol should be avoided. Saline aperients and small doses of iodide of potassium should be administered, but the latter should be increased with caution so as to avoid flooding the blood with a soluble lead salt. The treatment of the constipation, colic, paralytic and other nervous lesions will be found in their appropriate places.

V. **Incipient Tuberculosis** is generally attended by anæmia, pallor, weakness, and loss of flesh. The anæmia is often very marked, and if a young anæmic patient is not amenable to treatment by iron latent tuberculosis should always be suspected (Trousseau). The disease may be entirely latent in the sense of being unattended by any physical signs in the lungs or elsewhere. Reference has previously been made to a case of tuberculous meningitis (footnote, p. 641) in which for the first nine weeks the only signs or symptoms were anæmia, headache, and intermittent pyrexia. It is useful to remember that a tuberculous process, no matter where it is situated, is always attended by pyrexia of an intermittent type, though this is extremely apt to be overlooked (§ 382). Enquiry should therefore be made for sweatings or "chills," careful temperature readings should be procured, and the sputum and urine should be examined for the bacillus. The early diagnosis of tubercle in the lungs, meninges, kidneys, peritoneum, and other parts have been given in their appropriate places. Early spinal caries may also be overlooked, and the only symptoms present may be slight pain in the hypogastrium.

VI. Incipient or **Latent Carcinoma** and sarcoma are also attended by pallor, weakness, and emaciation; they form the essential parts of cancerous cachexia. The pallor does not yield to iron. Emaciation is, however, usually the most constant and most prominent feature, and therefore malignant disease will be considered fully under that symptom (§ 416). I have twice mistaken cases of scirrhus of the pylorus for examples of primary anæmia.[1] They were cases in which the pyloric end of the stomach was drawn up under the liver and entirely escaped detection during life. The diagnosis is all the more difficult when it occurs, as it did in these cases, without vomiting, and in comparatively young women, aged 28 and 35 respectively. There may be hæmic murmurs, and on rare occasions intermitting pyrexia. Another case of anæmia which came under my notice for a severe persistent neuralgia of the third sacral nerve, lasting many months, eventually proved to be carcinoma of the prostate. Repeated careful examination should be made of all the abdominal and pelvic organs.

VII. **Dyspepsia, Constipation, Colitis,** and various other disorders of the **alimentary canal** frequently come under our notice for anæmic pallor. Indeed, dyspepsia and confinement indoors are perhaps the commonest causes of pallor among hospital out-patients, and it must not be forgotten that decaying teeth associated with pyorrhœa alveolaris may, as Dr. William Hunter[2] has shown, be a potent cause of intense anæmia. Deficient food, and particularly deficient nitrogenous food, may also act in a lesser degree. In dyspeptics the pallor is of a peculiar kind in which the skin loses its lustre and may even be scurfy, and there are dark rings under the eyes. In colitis and other intestinal affections the same may be observed, and it is wonderful what an improvement in the patient's appearance is made after a course of treatment by salicylate of bismuth, naphthol, or other intestinal antiseptics, combined with purgatives. Many cases in this group are auto-toxæmias leading to deficient production or increased destruction of hæmoglobin, or both.

VIII. **Aortic valvular disease** often presents a sallowness which may be mistaken for the pallor of anæmia. The patient is usually an adult at or past middle life. In aortic regurgitation the cardiac murmur and the pulse (§ 50) are sufficiently characteristic for the detection of the disease on examination. But in aortic obstruction the most experienced auscultators may fail to detect or may misinterpret the signs of the lesion.

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[1] *Clinical Journal*, Feb. 13, 1895, vol. v., p. 251.

[2] *The Lancet*, 1901 and 1902.

IX. **Chronic Renal Disease** is sometimes accompanied by a pallor which may readily be mistaken for primary anæmia. This is especially the case in chronic parenchymatous nephritis, which is apt to affect young people. The pallor, however, is of an ivory whiteness, is usually accompanied by a certain amount of dropsy, and the urine reveals a definite amount of albumin and tube casts. Chronic interstitial nephritis is mostly met with in older people; it is generally attended by sallowness, but is mentioned below under progressive asthenia, which is its more constant and striking symptom.

X. **Cirrhosis of the Liver** may be attended by an anæmic pallor; but it is usually attended also by dilatation of the venous capillaries in the face, which are very characteristic. The signs and symptoms of this disease may be very obscure.

Certain other secondary anæmias are readily detected by their history, viz., HÆMORRHAGE or long continued drain on the system; CHRONIC SUPPURATION or other septic processes; RHEUMATIC and other FEBRILE CONDITIONS.

XI. Anæmia may be due to **hæmorrhage**—profuse and sudden or small and frequent; hyperlactation, profuse chronic diarrhœa and other debilitating conditions. Such cases are generally discoverable by a history of the cause, such as menorrhagia, post-partum or ante-partum hæmorrhage, bleeding piles, melæna, recurrent epistaxis, hæmatemesis, hæmoptysis, hæmaturia, etc. The treatment resolves itself into attacking the cause and applying the same measures as for chlorosis. But in severe cases of post-hæmorrhagic and other forms of intense anæmia, and whenever collapse is present, transfusion or infusion may be called for.

§ **407.** TRANSFUSION OF BLOOD OR INFUSION OF SALINE SOLUTION may be indicated in the presence of shock, collapse, or intense anæmia, arising in three groups of conditions: (1) Acute anæmia, resulting from abortion, post-partum and other causes of uterine hæmorrhage, gastric or intestinal ulceration, operations attended by profuse bleeding, or internal hæmorrhage after abdominal injuries. (2) Collapse consequent upon surgical operations, cholera, or severe diarrhœa due to any cause. (3) Blood poisoning, *e.g.*, in uræmia, puerperal eclampsia, diabetic coma, or poisoning by carbon monoxide, carbon dioxide, illuminating gas, carbolic acid, strychnine or phosphorus. *Transfusion of blood* from one person to another is at the present time not so much employed as in the past. It is indicated where the nutritive value of the blood is required. It is described in surgical text-books. *Infusion of saline solution* can be much more rapidly and easily performed, and skilled assistance is not necessary. As a mere diluent or to make up the volume of the blood it is of great value, and has almost entirely superseded the transfusion of blood. In cases of blood poisoning (group 3 above) venesection should be performed to eliminate the poison before the saline solution is administered. Sodium chloride (a neutral salt) is used to bring the specific gravity of the fluid to be injected up to that of the blood plasma. The normal saline solution (0·75 per cent.) is prepared very readily by dissolving 1 drachm (or to be exact 66 grains) of common salt in a pint of *boiled* water. The infusion may be made (1) into a vein (which is only done in very urgent circumstances); (2) into the subcutaneous cellular tissue of the thighs, anterior abdominal wall, below the clavicle, or mammæ; or (3) into the rectum. For injecting the solution into the subcutaneous tissue an ordinary exploring needle connected to a rubber tube 5 to 7 feet long, with a glass funnel or a douche-can such as that used in midwifery operations—all rendered aseptic—and a thermometer, complete the necessary apparatus. The needle is inserted after cleansing the skin, and the operator standing on a chair pours the solution (at a temperature of 99° or 100°) from a height of 5 to 7 feet above the needle slowly into the funnel. Three to six ounces may be injected at intervals of 10 or 15 minutes. If necessary, more than one puncture may be made. Usually ½ to ¾ pint is sufficient, but 2 or 3 pints may be needed. The puncture must afterwards be sealed with collodion. The danger of abscess or sloughing is very slight in young or middle-aged persons if the infusion be slowly done and not too much in one place, and if aseptic precautions are thoroughly carried out. To inject saline solution into the rectum an ordinary Higginson's syringe is employed. This method is simple yet often of great service, particularly in the collapse after operations.

XII. **Chronic Suppuration** (with or without amyloid disease) and other septic processes and causes of a prolonged drain on the albuminous materials of the blood are potent sources of anæmia. Prolonged lactation also may act in this way. Mention of the leucocytosis which is characteristic of suppuration or abscess has already been made (§ 399) but in addition the hæmoglobin and the red cells may be deficient.

XIII. **Post-febrile Anæmia**, associated nearly always with weakness and emaciation, may ensue after rheumatic fever, enteric fever, varicella, malaria, and indeed after any of the acute specific fevers.

XIV. Finally, in various conditions referred to in Group II. below (Emaciation), or Group III. (Debility), the anæmic pallor may be the symptom which first attracts our notice, for these 3 important symptoms are so often associated. Early myxœdema (§ 420), is one of these, and the puffiness of the eyes, the failing memory, loss of hair, and bodily weakness may for a time escape observation, or be attributed to other causes.[1]

Rarer Causes of Anæmia.

The patient is pale *and anæmic; there is* ENLARGEMENT OF THE SPLEEN, *or the* LYMPHATIC GLANDS, *or both, and characteristic*

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[1] Cases of this kind are referred to by Dr. Charles Chapman, *The Lancet*, Sept. 30, 1899, p. 876.

CHANGES IN THE BLOOD—the disease is probably LEUKÆMIA, HODGKIN'S DISEASE, SPLENIC ANÆMIA, or a SEQUELA of MALARIA.

§ **408. I. Leukæmia** (syn. leucocythæmia, splenic leukæmia, lymphatic leukæmia) is a comparatively rare disease characterised by progressive anæmia, a large and persistent increase of white corpuscles, a slight diminution of the red cells, accompanied by enlargement of the spleen. There are two varieties of the disease—(i.) the *spleno-medullary* or myelogenous type (splenic leukæmia), and (ii.) *lymphatic* leukæmia. In the former the chief changes are in the spleen (which is enlarged) and the bone-marrow, and the blood contains myelocytes which are derived from the bone-marrow. In the lymphatic variety the chief changes are in the lymphatic glands, which are enlarged, and the blood contains an excess of lymphocytes derived from those glands. It is still an open question whether the latter is or is not related to lymphosarcoma and to Hodgkin's disease.

The *symptoms* are:—(1) Anæmia, with its concomitant symptoms such as breathlessness, enfeeblement, etc., which are generally the first symptoms to be complained of. In the early stages the pallor may not be very noticeable, but anæmia and the characteristic blood changes continue throughout, and in the advanced stages there is no disease which gives to a patient such an extremely pallid appearance. (2) Enlargement of the *spleen* occurs early in the disease, and the patient may even first come under our care complaining of abdominal swelling. The enlargement is less in the acute than in the chronic forms of the malady, when it may extend as far or farther than the umbilicus and weigh as much as 15 lbs. The enlargement is uniform and the consistence hard. It is due to a parenchymatous increase of all the tissues, though especially of the malpighian bodies. The liver also undergoes moderate enlargement in about two-thirds of the cases. The disease is accompanied by gastric disturbance, diarrhœa, and vomiting at irregular intervals. (3) In about one-third of the cases the lymphatic glands are enlarged; this feature constitutes the *lymphatic* variety of the disease. The thymus may also be enlarged, leading to dulness over the manubrium. In some cases all the adenoid tissue of the body is increased. (4) *Hæmorrhages* may occur from time to time from the nose or elsewhere, and into the skin or retina. (5) *Pyrexia* of an irregular type occurs in three-fourths of the cases, especially towards the end. (6) The urine generally shows an excess of *uric acid*.

(7) The *blood changes* are pathognomonic and vary in the spleno-medullary and the lymphatic varieties of the disease. In *both varieties* there is (i.) an increase of the white cells—1 to 5 or 10 red cells instead of 1 to 500 as in normal blood, and (ii.) a slight diminution in the number of the red cells. The blood changes in the *spleno-medullary* form of leukæmia are:—(i.) the presence of large mononucleated cells with neutrophil granules, which do not occur in normal blood nor in other forms of leucocytosis, and are regarded as special to this type of leukæmia. They are known as "marrow cells" or myelocytes, and constitute about 30 per cent. of the white corpuscles present. (ii.) The eosinophil cells are increased in number and may be larger than normal; (iii.) the normal polynuclear leucocytes may be slightly increased; (iv.) "mast cells" with coarse basophilic granules are present; (v.) nucleated red cells are

common and megaloblasts are found sometimes: (vi.) the blood platelets are often increased. The myelocytes and normal polynuclear cells form the bulk of the leucocytes present. In severe cases the blood may resemble pus under the microscope: the proportion of the white to the red corpuscles being 1 to 10, and sometimes even 1 to 1. In blood slides Charcot's crystals separate out and the hæmoglobin crystallizes readily. The blood changes in the *lymphatic variety* are:—(i.) The small mononuclear cells (the lymphocytes) are increased, and the most characteristic feature of the disease is that they form the bulk (sometimes as much as 99 per cent.) of the leucocytes present. In some cases the large instead of the small mononuclear cells are found to be increased. (ii.) The eosinophil and polynuclear cells are normal in number; (iii.) there are rarely any nucleated red corpuscles, and myelocytes are generally absent; and (iv.) the disproportion between the red and white cells is never so great as in the splenic variety.

The *diagnosis* is not difficult as a rule. Splenic leukæmia is the only form of anæmia accompanied by enlarged spleen and the changes in the blood described above. Lymphatic leukæmia clinically resembles Hodgkin's disease (*q.v.*), but has different blood changes.

TABLE OF THE DIAGNOSIS OF LEUKÆMIA.

	Spleen.	*Lymphatic glands.*	*Leading Blood changes.*
Splenic Leukæmia.	Spleen greatly increased.	Not usually enlarged.	Leucocytosis marked, chiefly due to myelocytosis. Eosinophil cells also increased. Red cells markedly deficient.
Lymphatic Leukæmia.	Spleen moderately increased.	Moderately enlarged.	Leucocytosis marked, due to increase of lymphocytes. Red cells show progressive anæmia.
Hodgkin's Disease.	Spleen slightly enlarged in $\frac{4}{5}$ of the cases.	Greatly enlarged.	Slight leucocytosis in some cases; moderate anæmia.
Splenic Anæmia.	Spleen greatly enlarged.	Not enlarged.	Leucopenia; marked anæmia.

The *prognosis* is very grave. The disease is progressive in character though the course is irregular, with apparent restorations to health followed by relapses. Its average duration is 2 to 3 years, but its course may extend over 5 to 10 years, when it is of the chronic type. In a certain proportion of cases the type is acute. This especially occurs in young people and in the lymphatic variety, and the disease then runs a rapidly fatal course in 4 to 6 months or even less. Rarely the two varieties are combined. The complications are (i.) ascites and general dropsy due to heart failure; (ii.) intracranial hæmorrhages, or profuse hæmorrhages in other situations; and (iii.) severe diarrhœa and acute peritonitis.

Etiology. The disease is twice as frequent in males as in females. It usually comes on between the ages of 30 and 50, but may occur at any age. There is a history of malaria in a large proportion of cases, as high as three-fourths according to some authorities. Syphilis, rickets, rheumatism, traumatism and heredity have been supposed to play a part in the causation.

Treatment is of but little avail. In addition to general hygienic measures arsenic is the sovereign remedy, though it does little except hold the disease in check for a time. Cod-liver oil, quinine, and other tonics have been employed without much success. Oxygen inhalations have been suggested. Splenectomy is not justifiable.

Chloroma or "green cancer" is an anatomical term applied to a semi-malignant growth met with in the periosteum of the head and face and other places where lymphoid tissue is found. It is a rare disease, similar in its clinical symptoms to lymphatic leukæmia and lymphosarcoma, and is supposed to be pathologically a connecting link between them.

Dr. R. Hutchinson[1] says, "It would seem we have in lymphosarcoma, in chloroma, and in lymphatic leukæmia three closely allied processes exhibiting a descending scale of malignancy, but presenting many features in common, and in the case of chloroma and lymphatic leukæmia at all events possibly tending to pass into each other."

§ **409.** II. **Hodgkin's Disease** (syn. lymphadenoma) is a disease characterised by anæmia, progressive hyperplasia of the lymphatic glands, and sometimes lymphoid growths in the liver, spleen, kidney, and other organs. There are two forms of the glandular enlargement, soft and hard. In the former the glands are soft in consistence and somewhat enlarged; there is a proliferation of the endothelial cells, dilatation of blood vessels and lymph sinuses, while masses of lymphocytes crowd the lymph sinuses, and large multinuclear cells also occur. In the hard variety, which is usually a more advanced form, the glands are much enlarged and hard in consistence; and there is a great increase in the fibrous tissue which takes place at the expense of the other cells.

Symptoms. (1) Sometimes debility and anæmia are the first symptoms but more frequently enlargement of the lymphatic glands of the neck, axillæ, or groins first attracts attention. The enlargement of the cervical or other single group of glands may precede that of any other glands for a considerable time, even for a few years, but usually the extension to other glands is more rapid. In chronic forms of Hodgkin's disease the glands are hard, separate and moveable under the skin; but in the acuter forms the glands feel soft and great enlargement and suppuration may occur. For accurate diagnosis a gland should be excised under local anæsthesia and submitted to an expert.[2] In Hodgkin's disease the glands readily become the seat of secondary infection; hence probably the variation in the clinical symptoms and course. (2) Pressure effects occur when the deep glands, *e.g.*, in the thorax, become involved, and occasionally they are the first to enlarge. The pressure symptoms are described on p. 57. Bronzing of the skin may arise as a consequence of pressure on the solar plexus. (3) Irregular paroxysms of intermittent pyrexia occur at intervals of a few days or a few weeks, and these attacks coincide with a paroxysmal enlargement of the lymphatic glands. (4) The spleen enlarges as the glands enlarge, but the enlargement is never very great; usually the edge can just be felt below the costal margin. The liver also enlarges in most cases. (5) The blood changes are not characteristic: they consist chiefly of diminution of the hæmoglobin and the number of red corpuscles with poikilocytosis in the later stages. Slight leucocytosis may occur towards the end. (6) The constitutional symptoms, anæmia, and languor increase, and in the later stages may become extreme, with the concomitant symptoms of emaciation and a marked tendency to hæmorrhages (as is usual in all profound anæmia).

Diagnosis. Clinically, Hodgkin's disease and the *lymphatic variety of leukæmia* are alike, but an examination of the blood at once reveals the difference. The blood in Hodgkin's disease shows only a diminution of the hæmoglobin and the red cells, and in the later stages only a slight degree of polynuclear leucocytosis; in leukæmia there is always a *marked and characteristic increase in the number of lymphocytes* throughout, and usually an increase in the total number of leucocytes. *Lymphosarcoma* has been considered on the Continent to be akin to Hodgkin's disease. In lymphosarcoma there is a degree of lymphocytosis present in the blood, but the growth, though primarily involving the lymphatic glands, invades the surrounding tissues and thus reveals its malignancy. *Lymphoma* cannot be diagnosed from the chronic cases of Hodgkin's disease, when only one group of glands is enlarged, and the diagnosis from *tuberculous adenitis* is often difficult. Tubercle is more common at an earlier age; the glands tend to be matted together and to caseate or suppurate. The tuberculin test, if positive, proves the presence of tubercle, but it cannot decide whether the morbid process in a given case was tuberculous at the onset. *Syphilitic* glandular enlargement is preceded by the appearance of a chancre; the glands are very hard, and tend to disappear rather than to spread.

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[1] *The Lancet*, May 21, 1904, p. 1408.

[2] Dr. Dorothy Reed has shown that the glands show characteristic changes: (i.) proliferation of the endothelial cells and excess of lymphocytes filling the lymph sinuses, and later (ii.) excess of fibrous tissue. ("John Hopkins' Hospital Reports," 1902, vol. x., p. 133.)

Prognosis. Hodgkin's disease usually runs a slow chronic course, months or even years elapsing before extension from one group of glands to another. On the other hand the disease may run an acute course, all the glands enlarging within a few months. Cases are reported to have recovered or improved or remained stationary for a long period without special treatment, and chronic cases have entirely recovered under treatment. In severe cases the anæmia and emaciation are marked and death occurs from exhaustion, or with delirium and coma. Complications such as pneumonia, pleural effusion, pressure on the bronchi or trachea may also cause death. Difficulty in swallowing and deafness may arise from overgrowth of the adenoid tissue in the pharynx or thorax, and lymphoid growths in the walls of the stomach lead to dyspepsia and vomiting.

Of the *ætiology* little is known. The disease usually arises in the first half of life. Among Sir William Gowers' 100 cases 30 were under 20 years old, 34 between 20 and 40, and 36 over 40. It is three times as frequent in men as in women. An infective origin has been suspected. In some cases the disease has been attributed to an attenuated tuberculous infection. In other cases a local cause of irritation, such as nasal catarrh or a bad tooth, has led to a local enlargement of a group of glands which was followed later by a generalised lymphadenomatous enlargement.

Treatment. Local chronic groups of glands should be removed. Recoveries have been reported with the use of arsenic. It should be administered for months at a time in gradually increasing doses until tolerance is reached. Iodides are useless. Phosphorus should be tried if arsenic is ill borne. Tonics, cod liver oil and all other means to keep up the patient's strength are useful.

§ **410**. III. **Splenic Anæmia** is a rare disease, the characters of which have been thus summarised by Dr. H. D. Rolleston[1]: (1) splenic enlargement which cannot be connected with any recognised cause; (2) absence of any enlargement of the lymphatic glands; (3) anæmia of a type midway between secondary anæmia and chlorosis; (4) leucopenia, or at most no leucocytosis; (5) an extremely prolonged course lasting years; and (6) a tendency to hæmorrhages, especially gastro-intestinal, from time to time. The patient may come under observation during the *early* stage of the disease when anæmia with its concurrent symptoms is complained of. In the *second* stage there is enlargement of the spleen accompanied by attacks of pain during periods of enlargement of the organ. The spleen in this disease attains an enormous size, almost as great as that which occurs in leukæmia. The enlargement is due to fibrosis of the organ, with atrophy of the malpighian bodies. In some cases the splenic enlargement appears to precede the anæmia, and the patient may not seek advice until his spleen has reached the umbilicus. As the disease progresses there is loss of strength without emaciation, accompanied by gastric disturbance and a tendency to hæmorrhages. Sometimes there is moderate enlargement of the liver. Pyrexia is present during the active stages of the disease. The chief distinction between this disease and splenic leukæmia is in the blood changes and particularly in the absence of leucocytosis in splenic anæmia. The blood shows a diminution of the number of the red cells, with a diminution of the hæmoglobin corresponding to the chlorotic type of blood. Poikilocytosis may be present. There is usually some diminution of the leucocytes also (leucopenia). In the *third* stage of the disease all the symptoms are aggravated, and in a few cases of the disease the so-called "Banti's disease" supervenes, with fatal termination. The name Banti's disease has been given to a group of symptoms comprising cirrhosis of the liver, jaundice and ascites, consequent on a history of splenic anæmia as described above.

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[1] *The Lancet*, August 8, 1903, p. 401.

The *diagnosis* from most forms of *secondary anæmia* is effected by the great enlargement of the spleen; and from *leukæmia* by the characteristic blood changes in that disease (§ 408). *Pernicious anæmia* is rarely associated with an enlarged spleen, but difficulty may arise when the blood changes in a severe case of splenic anæmia resemble those of pernicious anæmia. The chief practical difficulty lies in diagnosing the disease from *cirrhosis of the liver* with accompanying enlargement of the spleen. If the red corpuscles and the hæmoglobin rapidly increase under treatment by iron, it is improbable that the condition is due to splenic anæmia. Banti's disease may be almost impossible to diagnose from cirrhosis of the liver unless a history including the blood changes of previous years can be obtained. Many cases regarded during life as splenic anæmia are found after death to be due to visceral syphilis, cirrhosis of the liver, or thrombosis of the portal vein.

Prognosis. The disease is a chronic progressive disorder. It used to be said that death occurred in six months to two years, but it is now known that cases may live ten, twelve, or even twenty years after the commencement of the disease. Death takes place by asthenia, occasionally by syncope or hæmorrhage.

Etiology. Men are more often affected by this disease than women, and it occurs mostly in adult life. The cause is unknown.

The *treatment* is the same as that of other anæmias. Arsenic is the most efficacious drug. Splenectomy may prolong life.

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[1] Murrell, *The Lancet*, April 26, 1902, p. 1177.

The patient is very pale *and anæmic, and there are or have been* SORENESS *of the* GUMS, PURPURIC SPOTS *or other hæmorrhagic lesions*:—the disease is probably SCORBUTUS.

§ 411. IV. **Scurvy** (syn. scorbutus) is a constitutional disease due to deficiency of fresh food, animal and vegetable, attended by extreme debility and anæmia, sponginess of the gums, and hæmorrhages. The disease is only rarely met with now, but used to be the scourge of the British navy, until the introduction of lime juice as a prophylactic, which now all ships are obliged to carry.

The *symptoms* are of insidious onset and consist of (i.) progressive debility and anæmia, with mental depression and headache, but no pyrexia. Palpitation, hæmic murmur, syncopal attacks, and other symptoms of anæmia develop. Pains in the back and limbs are usually complained of early. The urine is scanty and highly acid, and may contain albumin. (ii.) The gums become spongy, swollen, and bleed readily. Sloughing may follow, and the teeth become loosened; the breath is very offensive. Constipation is usual, but diarrhœa with blood may occur later on. (iii.) A characteristic eruption appears, consisting of purpuric spots and swellings of brawny consistence found about the flexures of the joints, especially the popliteal space. These swellings are due to hæmorrhages into or beneath the skin; if the former they are purple, but if beneath the skin the colour may be pale. Swellings may also occur under the periosteum of the bones of the legs. Epistaxis often occurs, but hæmorrhage from other mucous membranes is not common except in severe cases. (iv.) Death may ensue either from syncope, asthenia, or complications. Among the latter may be mentioned sanguineous effusion into the pleura or meninges, pneumonia, and sloughing of the skin.

Diagnosis. The diagnosis of scurvy from other causes of *purpuric* eruption is afforded by the condition of the gums, and the hard brawny swellings, which are peculiar to scurvy, and also by the degree of prostration present. Slighter cases are, however, very difficult to diagnose, as similar symptoms may be seen with purpura. Rapid improvement with suitable diet favours a diagnosis of scurvy. *Syphilitic nodes* on the tibiæ accompanied by cachexia should be carefully differentiated from scurvy, because mercury is so injurious in the latter disease. In *pernicious anæmia* there is a longer history, and blood changes are present.

Prognosis. As a rule this is good if the cause be discovered, and removed. Unfavourable symptoms are severe dyspnœa, syncope, scanty urine, and elevations of temperature. Convulsions, hemiplegia, and other cerebral symptoms follow intracranial hæmorrhage. The outlook is grave when dysentery complicates the disease. Necrosis of the jaw or other bones is rare.

Etiology. The chief cause is an absence of fresh vegetable from the dietary, though this will not produce scurvy if fresh meat is available. It is the combination of salted or tainted meat and the absence of fresh vegetable which leads to scurvy. It has occurred in well-to-do women who live on tea and bread-and-butter. It appears that organic salts are more unstable than others, and thus potassium, for example, if combined with an organic salt, will more readily reach the tissues and there be converted into a carbonate, than if it be taken in the form of a chloride or a phosphate, which are more stable salts. In fruits and vegetables which have organic salts, therefore, the mineral constituents more easily reach the tissues than in animal foods, which have inorganic salts.

Treatment consists in giving a liberal diet, with fresh vegetables, the juice of 2 lemons daily, fruits, light wines, claret, tea, and cider. Scraped raw meat and milk are good for severe cases. For the mouth give a gargle of pot. chlor. or Condy's fluid, or pencil the gums with a strong solution of silver nitrate. Give bismuth and opium for diarrhœa, and avoid foods which would irritate such complications as dysentery.

The patient is pale *and liable to* UNCONTROLLABLE BLEEDINGS, *with little or no cause*—the morbid condition is probably HÆMOPHILIA.

§ **412.** V. **Hæmophilia** is a hereditary disease characterised by a constitutional tendency to uncontrollable hæmorrhage without sufficient cause.

The *symptoms* are divided into three sets:—(1) hæmorrhages from mucous membranes, or after some slight injury from the skin. Nothing abnormal may be noted in a subject of hæmophilia until he has a tooth extracted or a trifling abrasion, when uncontrollable bleeding due to capillary oozing sets in and lasts for hours or days. When the bleeding occurs from a mucous surface large blood tumours may form as the blood coagulates. (2) Interstitial hæmorrhages occur spontaneously or after injury in the form of petechiæ or hæmatomata. (3) Affections of the joints, especially the knees and elbows, are met with, and three stages are described—(i.) recurrent hæmarthroses or effusions of blood into the joints, of acute onset, sometimes attended by pyrexia; (ii.) reactionary synovitis, and (iii.) cicatrisation which may lead to permanent deformity.

Diagnosis. A single severe hæmorrhage does not warrant a diagnosis of hæmophilia, but recurrent hæmorrhages with slight cause are characteristic. The family history of a tendency to bleeding is important. The joint affections are diagnosed by the presence or history of other signs of hæmophilia.

Prognosis. The disease usually becomes evident during the first few years of life, and as a rule tends to be less troublesome as life advances, disappearing about 30 or 40. Great anæmia occurs from excessive bleeding, and life has been lost after trivial operations such as the extraction of a tooth, or circumcision.

Etiology. Hæmophilia occurs in families for generations. It is met with in males, in the proportion of 13 to 1 female, but the diathesis is transmitted through the female who herself may remain unaffected. The cause of the condition is unknown; the blood vessels in some cases have been found to be thin, and A. E. Wright has found the blood coagulability is retarded as long as 20 to 45 minutes.

Treatment. Males in a bleeder's family should be guarded from any injury or operation. The daughters should not marry, for though they themselves are not endangered by parturition their sons will probably be bleeders, and their daughters will pass on the same tendency in turn to their offspring. When bleeding occurs, rest is essential, and styptics are applied. Internally adrenalin chloride, calcium chloride, iron perchloride and ergot have done good.

VI. **Addison's Disease** and other maladies mentioned in **Groups II.** and **III.** (below), occasionally come under our notice for anæmia.

There is **pallor** *of the skin and the patient has* BEEN ABROAD—enquiry should be made for MALARIA, CHRONIC DYSENTERY, PARASITES, or other TROPICAL DISEASES.

VII. Various **tropical diseases** and other pyrexial conditions rarely if ever seen in England, are attended by intense anæmia.

a. *Malarial anæmia* is usually accompanied by pigment changes around the eyes and other parts of the body. The history here is of course our first clue to diagnosis. The earthy pallor and the enlargement of the spleen (ague cake) are very characteristic.

b. *Dengue, beri-beri, dysentery* and other fevers unknown in this country are accompanied and followed by anæmia. In the two first named there is much general weakness and some emaciation, and in the third diarrhœa.

c. In Egypt and other countries, cases which used to be considered as idiopathic anæmia are now known to be due to *intestinal* and *other parasites*. The *ankylostomum duodenale* and *bothriocephalus latus* may be recognised by the presence in the fæces of ova or segments, and when the patient recovers under large doses of thymol it is probable that he has had ankylostomum duodenale. A widespread epidemic of ankylostomiasis occurring among the men employed in the Dolcoath mines, Cornwall, has recently been reported, of the same kind as that occurring during the construction of the St. Gothard Tunnel in 1880. *Bilharzia hæmatobia*, endemic in Egypt and elsewhere, causes anæmia and hæmaturia (p. 515). The *filaria sanguinis hominis* (see chyluria) and *strongylus gigas* (see melæna) also give rise to anæmia.

Distoma Pulmonale (syn. Distoma Ringeri) is a parasite found in the lungs, liver, testes, peritoneum, and brain. It is met with in Formosa (being present according to Manson in 15 per cent. of the inhabitants), China, and other tropical countries, where it gives rise to endemic hæmoptysis. It measures 8 to 10 mm. in length by 4 to 6 in breadth. It is reddish brown in colour, oval in form. In the lung, its favourite habitat, it gives rise to chronic cough without physical signs, rusty sputum, and irregular attacks of hæmoptysis, accompanied by intense anæmia.

§ 413. Anæmia in Children. All the forms of anæmia above described may occur in children under 14, and are produced by the same causes which affect adults, but they occur in a very different order of frequency, and present certain marked differences. *a.* The spleen tends to become enlarged in all forms of anæmia in children, but is markedly so in (1) splenic anæmia infantum, (2) lymphatic and myelogenous leukæmia, and (3) Hodgkin's disease. *b.* The blood changes also differ considerably from the blood changes met with in the same diseases in adults. In infancy and childhood slight causes lead to blood alterations of a marked type which, if occurring in an adult, would signify severe disease.[1] *c.* In secondary anæmias in children, as in adults, a diminution of the hæmoglobin is the earliest change, but there are important differences. In childhood (1) the number of red corpuscles is reduced at a comparatively early stage and new ones enter the blood in a half-formed condition—poikilocytosis and nucleated red cells—and (2) leucocytosis occurs more readily, chiefly of the mononuclear cells (small and large lymphocytes) owing to the activity of the adenoid tissue in children. Adenoid tissue is very active and plentiful in children, and hence in childhood an increase of the lymphocytes (which are supposed to come from the adenoid tissue and lymphatic glands) is more common than an increase of the polynuclear neutrophil and other white cells (which are supposed to come from the bone-marrow). Thus in children among the leucocytic diseases lymphadenoma, lymphatic leukæmia and lymphosarcoma are more common than spleno-myelogenous leukæmia.

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[1] See Dr. Robert Hutchison, "Disorders of the Blood in Early Life," *The Lancet*, May 7, 1904.

I. PRIMARY ANÆMIAS. *Chlorosis* is hard to diagnose with certainty in a child, but it may occur. *Pernicious anæmia* in childhood (which can only be recognised in them by the deposit of iron in the liver and fatty degeneration) is so rare that some authorities deny its existence. But blood changes with poikilocytosis and nucleated red cells such as occur in the pernicious anæmia of adults may in children be met with in many other anæmias without any grave significance attaching.

II. The chief causes of SECONDARY ANÆMIA in children are *defective nutrition*, the *acute specific fevers*, *acute rheumatism*, *prolonged suppuration*, *syphilis*, *tuberculosis*, and *chronic diarrhœa*. Secondary anæmia frequently occurs in children who have had deficient proteid food. It is met with therefore in children who have been suckled too long, or have had only milk food at an age when they should have had proteid foods containing iron.

In addition to the foregoing there are three ANÆMIAS SPECIAL TO CHILDREN—III. Infantile scurvy; IV. Splenic anæmia infantum; V. Congenital anæmia.

§ 414. III. **Infantile Scurvy** (syns. Scurvy rickets, Barlow's disease) used to be considered a variety of rickets, but it is now known that it need not necessarily be associated with rickets.

Symptoms. (1) The onset may be gradual. The child is noticed to become pale and in late stages profoundly anæmic. Muscular weakness becomes marked, but emaciation may be absent. The child cries when washed or dressed, screams if the legs are touched, and is very still when at rest. (2) Very soon an ill-defined swelling which visibly increases is seen along the tibia, just above the ankle. The swelling is not necessarily symmetrical, and is due to subperiosteal extravasation of blood. The legs appear as if paralysed, because the child keeps them everted and motionless, dreading the pain caused by movement. Sometimes the femurs also are affected and there may be œdema of the dorsum of the foot. The arms and scapulæ are next affected; the ribs, skull and face rarely so. The joints are free. (3) Swellings may occur in the muscles, resembling abscesses, but there is no redness or fluctuation. (4) Sponginess of the gums develops later on, and petechiæ and internal hæmorrhages may occur as in adult scurvy. (5) Rare symptoms are albuminuria and hæmaturia; and proptosis from hæmorrhage into the orbital periosteum. The *temperature* is normal except after large or recent hæmorrhages, when it may rise to 100° or 102° for a few days.

Diagnosis. Scurvy rickets may often be mistaken for *rheumatism*, but whereas in rheumatism the joints are affected, in scurvy they are free. *Infantile paralysis* has no swelling or tenderness of the limbs. *Abscesses*, *nephritis*, and *stomatitis* have no signs of scurvy. *Syphilitic* pseudo-paralysis has crepitation and pain on moving the limb, due to separation of the cartilage from the diaphysis.

Prognosis. On the whole this is favourable. Under treatment recovery is rapid, and the child may be well in three weeks. If the patient is seen at a late stage, or if from failure to diagnose the disease the diet is not altered, death occurs from syncope or complications such as diarrhœa, bronchitis and pneumonia or any of the acute specific fevers.

Etiology. The disease affects children of 6 to 18 months usually, and is due entirely to defective diet, *i.e.*, absence of fresh food. It occurs particularly in infants fed only with proprietary foods, boiled, sterilised or condensed milk.

Treatment. The prophylactic treatment consists in the observance of a few simple dietetic rules. Fresh unboiled milk must be used, or the milk must only be scalded (brought to the boiling point for a second). Peptonised milk and artificial foods should never be used for longer than a few weeks at a time. Remedial treatment consists in the administration of fresh milk. Steamed potato, rubbed through a sieve and beaten up with milk to the consistence of thick cream, should be given in doses of ʒj. to ℥j.

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with each bottle. A few ounces of beef-tea, in which carrots have been boiled, and then strained out, may be taken twice daily. Raw meat juice is excellent. For children of a year old, grape, orange, lemon, and baked apple juice, with potato pulp and raw meat juice should be given. *Local* treatment consists in wrapping the limb in cotton wool and preventing movement.

§ 415. IV. **Splenic Anæmia of Children** (syns. anæmia infantum pseudoleukæmica, von Jaksch's disease, anæmia splenica infettiva dei bambini) occurs in children from six months to two years of age, and is characterised by anæmia and leucocytosis and enlargement of the spleen. The splenic anæmia of adults is not the same disease.

Symptoms. (i.) Pallor due to anæmia of insidious onset, sometimes preceded, sometimes followed by (ii.) enlargement of the spleen, which may attain a great size. Attacks of pain may occur due to perisplenitis. (iii.) The liver is moderately enlarged, and in some cases the glands also. (iv.) There is irregular pyrexia and gastro-intestinal disturbance. (v.) The patient may remain plump throughout, but in severe cases usually becomes greatly emaciated. (vi.) In serious cases hæmorrhages occur from the mucous membranes and into the skin. (vii.) The blood changes are characteristic—the hæmoglobin is diminished, the number of the red corpuscles is reduced usually to two or three million, and a slight degree of poikilocytosis is present, together with nucleated red corpuscles. Leucocytosis may be absent in the earlier stages, but always marked in the later stages. The polymorphous character of the leucocytosis is a diagnostic feature. Myelocytes, lymphocytes, large mononuclear cells, and many transitional forms are seen, the transitional forms rendering a differential count impossible.

The *diagnosis* is difficult only in the early stages. In both *syphilis* and *rickets* we often meet with anæmia, enlargement of the spleen and of the liver, but the spleen never attains the same size, and the blood changes are never so marked as in splenic anæmia of infants. In children severe *secondary anæmia* may present leucocytosis, but the polymorphism of the leucocytes is not found. *Splenic leukæmia* rarely, if ever, occurs in children, and the leucocytosis has different features. The diagnosis of splenic anæmia of children depends on different features at different stages; and it would appear, from the numerous synonyms above mentioned, that this disease has been described by various observers, under different names, according to the stage under observation. In the early stage the changes in the red corpuscles are prominent, resembling those in severe anæmia; in the later stages the leucocytosis becomes more noticeable, hence the name pseudo-leukæmia.

The *prognosis* is good. The course is short and recovery usually complete, but cases relapse under bad hygienic conditions. Hæmorrhages and petechial eruptions are serious symptoms. The lower the number of red, and the higher the number of white corpuscles the graver is the prognosis. Death occurs from exhaustion or intercurrent diseases.

The *etiology* is obscure; the disease is supposed to be due to some specific gastro-intestinal toxin. It is often associated with rickets. No connection has been traced between this disease and syphilis.

The *treatment* consists in remedying the hygienic conditions. Fresh air is essential and good food, such as yolk of egg, raw meat juice, potatoes and bone-marrow should be given. Of drugs, iron and arsenic are the best; cod-liver oil and malt are useful adjuncts.

V. **Congenital Anæmia** occurs occasionally. The causes are obscure. No iron is obtained during the period of suckling, but a child is born with a store of iron in the liver (Bunge), and it may be assumed that this store of iron has for some reason been deficient. Sometimes there is a history of icterus after birth, as if there had been an abnormal amount of blood destruction at that time. In other cases the bone-marrow has been found to be defective. The condition is very apt to be confused with congenital syphilis.

GROUP II. EMACIATION.

Wasting is a common sequence of nearly all acute and many chronic diseases, but when it is the leading or only symptom the following morbid conditions should be borne in mind. The fallacies have been referred to in § 394.

I. Malignant disease.

II. Defective feeding and digestive disorders, colitis, intestinal adhesions or stenosis, and other obscure intestinal conditions.

III. Tuberculosis, diabetes mellitus, diabetes insipidus, chronic Bright's disease, syphilis, and other diseases of Groups I. and III.

IV. Diseases of the pancreas.

V. Various nerve conditions.

Marasmus in children may be caused by—defective feeding, diarrhœa, constipation, persistent vomiting, hereditary syphilis, rickets, tabes mesenterica, and pulmonary tuberculosis.

Emaciation in the last third of life is suspicious of carcinoma; in the middle third of life diseases in II. and III. above; and in the first third, tuberculosis.

§ 416. I. **Malignant Disease** (carcinoma and sarcoma) is a cause of emaciation which should be ever present in the mind when the patient is at or past middle age. There are two anatomical varieties—carcinoma (cancer proper) and sarcoma. The essential feature common to them both is that they tend to recur after removal, to invade the parts around and to reproduce themselves in distant parts; this is the clinical meaning of the word malignant. Certain localities are much more prone to primary deposits than others, and in cases of latent malignant disease it is important to remember the places in which primary carcinoma and sarcoma may occur. The commonest seats for *primary carcinoma* are the skin around the mucous orifices, the tongue, œsophagus, stomach, colon, rectum, mamma, uterus, penis, and testis. In these regions secondary carcinoma is almost unknown, unless by direct infiltrating invasion. *Sarcomata* are rarely found primarily in these situations, but are prone to start in the peritoneum, the fascia, the bones, the corium, ovary, kidney in children, brain, spinal cord, retina, and in the fibrous structures of the muscles, breast, and testicle. Different varieties vary considerably in their malignancy and rate of growth. The most rapidly growing of all is the round-celled sarcoma, the next in order being the spindle-celled sarcoma, the myeloid sarcoma (*e.g.*, epulis) being of slow growth, and rarely producing secondary tumours. Melanotic sarcoma has a great tendency to reproduction in distant parts. Among the carcinomata the softer and encephaloid varieties are more rapid and malignant than the harder and scirrhous varieties. Any tissue or organ of the body may be involved either by continuity or along the lymphatics (in the case of carcinoma) or along the veins (in sarcoma).

Symptoms. The symptoms of malignant disease necessarily vary with the situation, and under the heading of tumours or growths the diagnosis has been previously dealt with (*e.g.*, the abdomen, § 189, the chest, §§ 57 and 103). We are here concerned with a general review of the symptoms. (1) There is loss of weight quite early in the disease, sometimes long before any local signs can be detected. This is accompanied by a typical cachexia, *i.e.*, an appearance of illness, in which the skin assumes an ashy or sallow hue. The sallowness of the skin may be so marked as to be with difficulty distinguishable from jaundice, or even Addison's disease. (2) The age of the patient is advanced in carcinoma, young in sarcoma. The 4 classical signs of cancer are—pain, swelling, offensive discharge, and hæmorrhage. (3) Pain at the seat of growth is often complained of, especially in rapidly growing varieties, or when they occur in tense parts. (4) In accessible situations a thickening, swelling, or tumour may be detected, which is usually hard, nodular, and apt to fix and infiltrate the surrounding parts. Some sarcomata are soft and pulsating. (5) Whenever the growth involves a mucous or epidermal surface there is an offensive pink or sero-sanguineous discharge, *e.g.*, from the vagina. (6) In like manner hæmorrhage may occur and take the form either of metrorrhagia, coffee-ground vomiting, or melæna; and when the disease involves the pleura or peritoneum the *effused fluid will be blood-stained.* (7) In carcinoma the neighbouring lymphatic glands become enlarged and palpable. (8) The rate of growth is rapid, though it varies widely in different forms and localities. Scirrhous infiltration of orifices may only reach the thickness of half an inch in 6 to 12 months, and the patient may live 2 years; but a round-celled sarcoma will reach the size of a hen's egg in a month or two and kill in six.

Diagnosis. Malignant disease may have to be diagnosed from all the other conditions which give rise to emaciation. A malignant nodule may have to be diagnosed from syphilitic gumma (compare, for instance, syphilis of the tongue, skin, etc.), but the latter is usually attended by less pain and constitutional disturbance, and is amenable to anti-syphilitic treatment. Cases of sarcoma are in rare instances attended by pyrexia, four instances being recorded by Hilton Fagge and Pye Smith in their classical work on medicine.[1]

The *prognosis*, if the case is untreated, is always of the gravest kind, the course rarely lasting more than 1 or, at the outside, 2 years. A few cases of undoubted malignant disease have undergone spontaneous involution.[2] The prognosis largely depends upon the stage at which the true nature of the case is detected. On this depends very largely both the prospect of arrest or removal. In general terms the prognosis also depends on—(1) the position and accessibility of the growth, how far vital structures are involved, and whether it is on or near the surface; (2) the structure of the tumour (*vide supra*); and (3) the age of the patient, to some extent, for growth is more rapid in the young.

Etiology. (1) In carcinoma the age of the patient is nearly always over 40, though I have seen cases of scirrhus of the pylorus in persons aged 28 and 33. Sarcoma, on the other hand, may affect children or adults of any age. Sarcoma is the malignant growth of the kidney that is specially met with under the age of 9 more often than at any other time of life. (2) Sex has not much influence, but the great frequency with which cancer arises in the mammae and uterus gives to statistics a marked bias towards the female sex. (3) Heredity has always been regarded by most physicians as a predisposing cause of malignant disease, though it has been disputed by others. It is probable that malignant disease is hereditary in the same sense as tuberculosis, by offering a predisposition.

(4) *Infectivity.* The question is still under discussion whether cancer is due to infection from outside sources, to peculiarity of cell growth, or to the invasion of a low form of animal parasite or protozoon. The reproductive cells divide in a manner different to the somatic or body cells, and it has lately been shown that malignant cells divide after the manner of reproductive cells.[3] The infectivity of malignant disease rests mainly upon 4 grounds—(a) certain facts which appear to point to the appearance of cancer in individuals who occupy certain houses where cancer has previously occurred; (b) the clinical resemblance of a rare form of disease now known to be due to the ray-fungus (actinomycosis), to a malignant new growth, and a similar resemblance in structure between certain other fungoid diseases and malignant disease; and (c) the undoubted infectivity of artificially produced sarcoma in animals;[4] (d) a plasmodium has been found to cause tumours in certain cruciferous plants.

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[1] Fagge and Pye Smith's "Principles and Practice of Medicine," 4th edition, pp. 88, 89.

[2] See Clin. Soc. Trans., 1898—9, and elsewhere.

[3] A summary of the two latest theories on the origin of cancer is given in *The Lancet*, 1904, vol. ii., p. 467.

[4] E. Billingham Smith and J. W. Washbourne, "Infective Sarcoma in Dogs," *B. M. J.*, 1899, vol. ii., p. 1346.

The *treatment* of cancer until recently has consisted of (1) removal as early as possible; the earlier and more freely this is done the more hopeful the result. (2) In certain cases of malignant disease of the mamma removal of the ovaries has been attended by involution of the growth.[1] (3) The treatment of malignant growths (more especially of sarcomata) by the injection of mixed toxins (the virus of erysipelas and other pyogenic organisms mixed) was introduced a few years ago by Coley. A number of cases of sarcoma that had been given up as hopeless after repeated operations were reported by Mr. C. Mansell Moullin[2] as having completely recovered under this treatment; (4) more recently still the X-ray treatment has proved efficacious in mammary cancer.[3] It has also proved even still more efficacious when cancer has been met with on the surface of the body. But so far it has not produced any influence on the progress of the internal cancerous deposits.

II. **Defective Feeding and Digestive Disorders.** Although malignant disease or tubercle should always be remembered in obscure cases of emaciation in the old and young respectively, perhaps the commonest causes of slight loss of flesh met with in practice are defective feeding and digestive disorders. Digestive disorders may of course exist without any wasting, and if the latter be marked and the patient advanced in life it is always suggestive of cancer of the stomach, especially if there be loss of appetite. Defective feeding without digestive troubles, and particularly deficiency in the fats and carbo-hydrates, may without any digestive disorder be attended by emaciation. Defective teeth are a potent source both of digestive troubles and loss of flesh. Various INTESTINAL CONDITIONS are often attended by undue spareness of body. Among the latent causes of this may be mentioned obscure intestinal stenosis and catarrhal colitis, both of which may be overlooked for a considerable time. Severe diarrhœa is often followed by rapid wasting, especially in children. Incipient cirrhosis of the liver may also be remembered as a cause of emaciation.

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[1] Dr. S. E. Herman, *Lancet*, 1899, vol. i., p. 1031.

[2] *Clinical Journal*, April 6, 1898, p. 435.

[3] Discussion at the British Med. Assocn. Meeting (Surgical), 1902, and Clinical Society of London, May, 1904.

III. **Tuberculosis** often first makes itself known to us by a causeless loss of weight. DIABETES MELLITUS and DIABETES INSIPIDUS, CHRONIC BRIGHT'S DISEASE, and other diseases mentioned in Groups I. (*ante*) and III. (*post*) may first seek medical aid by reason of wasting. This is particularly so in diabetes, where the inconsistency of his ravenous appetite and constant thirst with loss of weight may even strike the patient. On the other hand, some cases of diabetes, especially those which occur later in life, are associated with a well-nourished if not a full habit of body. Syphilis, so frequently a cause of wasting in infancy, rarely causes much emaciation in the adult, though children affected with the hereditary disease grow up stunted, slight, and delicate. In latent tuberculosis the trunk and limbs may be wasted although the face be plump and rosy.

IV. Among the rarer causes of loss of flesh which should be remembered in obscure cases are—obscure visceral disease, and especially **disease of the pancreas.** Emaciation frequently accompanies Kala-azar and Beri-Beri.

V. Diseases of the **nervous system** may sometimes start with or present generalised wasting, such as bulbar paralysis, and the idiopathic myopathies (mostly met with in childhood), but they also usually present their proper symptoms.

§ 417. Marasmus in Children. Infants and children emaciate with almost any disorder and with surprising rapidity. A sudden attack of diarrhœa may give rise to loss of flesh in twenty-four hours.

The principal causes are 8 in number:—

(a) Those which occur chiefly under 2 years of age—(1) Defective food or feeding; (2) diarrhœa, or constipation; (3) persistent vomiting; (4) hereditary syphilis; and (5) rickets.

(b) Those which are met with chiefly after 2 years of age—(6) Tabes mesenterica (enlargement of the mesenteric glands) (7) pulmonary tuberculosis.

(1) DEFECTIVE FEEDING constitutes the commonest cause of emaciation amongst the children of the lower classes. Such children are always fretful, the bowels are irregular and often constipated, the stools instead of being the normal orange colour of infancy become either green, grey, or white and "chippy" with particles of undigested food. The error may consist either of over-feeding, under-feeding, or a defect in the quality of the food

or the time that it is administered. Over-feeding is perhaps more common than the reverse. Undiluted cow's milk is very indigestible, and mothers of all classes have a tendency to make their child's feeds too strong and to give them too frequently under the notion that it will make a child "grow strong." The mother's milk, when she is out of health, or when lactation has been too prolonged, may be of too poor a quality to afford adequate nutrition. Preserved milks and foods taken for too long a time without any fresh milk lead to scurvy-rickets (§ 414). By the plan which has recently been introduced, the art of infant feeding has been reduced to a science, and those who can afford this method can obtain full particulars from the Walker-Gordon laboratory. A suitable dietary, which is within the reach of all classes, has been given in § 214a, *ante.* A good way of giving cod-liver oil to children is to pour away the oil from a box of sardines and replace it by cod-liver oil. After standing for 24 hours the oil becomes flavoured with the sardines, and both can be given together to the child.

(2) DIARRHŒA or CONSTIPATION, either alone or alternating, are potent causes of wasting in infancy and childhood, and these are frequently due to dietetic errors or want of care and cleanliness in the nursery. The subject of infantile diarrhœa is fully discussed in § 220. Many cases of chronic diarrhœa in childhood will yield to mistura ricini calcis (F. 64). Chronic constipation will undoubtedly result in marasmus. In a family with which I was well acquainted the first two children died of marasmus associated with the most obstinate constipation, probably due to a chronic colitis; the case of the third child, which the mother stated exactly resembled the others in all particulars, was following the same fatal course until systematic treatment by mist. ricini calcis resulted ultimately in restoration to health.

(3) PERSISTENT VOMITING is another cause of wasting in childhood. Like the preceding it may be due to errors of diet, especially too frequent or over-feeding, or to gastro-intestinal catarrh. Careful dieting, lime water, and proper intervals between the feeds will cure most cases. The reflex and other causes of vomiting (§ 192) must be considered when simple treatment is unavailing. In intractable cases feeding by the nose has been

resorted to. Hypertrophied pylorus is a rare local cause of vomiting in infants.[1]

(4) HEREDITARY SYPHILIS is a recurrent cause of wasting in some families. It is generally accompanied by snuffles or skin lesions of some kind (§ 105). The manifestations of hereditary syphilis always appear during the first year of life, generally during the first six months.

(5) RICKETS (§ 146) may be accompanied by wasting, but, as Dr. Judson Bury[2] aptly remarks "fat rickets are commoner than lean rickets." This disorder is recognised by the characteristic bone changes, generalised sweating, and generalised tenderness. Rickets may appear at any time between the fourth to the eighteenth month of life; very rarely after two years of age.

§ 418. (6) **Tabes Mesenterica** is a wasting disorder occurring for the most part in children of 2 years and upwards due to tuberculosis of the mesenteric glands. The disorder is becoming less and less frequent as greater care is exercised in the cleanliness and asepsis of the milk.

Symptoms. The onset is very insidious and may extend over many months. Gradually the limbs and face become shrunken and there are anaemia, listlessness, vague attacks of pyrexia, and sometimes abdominal cramps. The leading physical sign is the enlarged abdomen, which is generally tympanitic on percussion. Sometimes the enlarged glands can be felt, but more frequently there are localised thickenings and masses which give a doughy feeling resembling that met with in chronic tuberculosis of the peritoneum. Ascites is rare, but there is a marked tendency to the formation of adhesions. Attacks of diarrhœa with offensive stools occur from time to time.

The *varieties* depend upon the extent to which the tuberculosis affects the peritoneum as well as the mesenteric glands. If the peritoneum is extensively affected the case resembles chronic tuberculous peritonitis (§ 177). There is no doubt that many cases of the latter disease are still called tabes mesenterica. Sometimes the disease runs a more acute course with pyrexia, and resembles enteric fever, from which it can only be differentiated with difficulty when a chart is procurable.

Diagnosis. In addition to the diseases just mentioned tabes mesenterica may have to be distinguished from the distension of the bowels due to improper feeding, in which there is generally no pyrexia, no resistant masses, and disappearance on regulating the diet. Rickets may be attended by a flabby or distended abdomen, but is not usually attended with so marked emaciation, and the characteristic rachitic changes in the skeleton differentiate it.

Prognosis. When once fully established the disease nearly always terminates fatally by progressive asthenia, though the course of the malady is apt to be irregular, with intervals of apparent recovery followed by relapses. Sometimes the glands undergo a fibroid change, and what appear to be the most unlikely cases recover. Among the untoward symptoms are, acute local pain and tenderness indicative of peritonitis, constant diarrhœa, indicating ulceration of the bowels, and the evidences of tubercle elsewhere. The complications are numerous—ulceration of the bowels attended by pyrexia and intractable diarrhœa, general tuberculosis, abscesses forming and bursting in various situations, such as into the peritoneal cavity or from the umbilicus, the latter forming a chronic fistula. Intestinal obstruction may result at any time from the formation of bands of adhesion.

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[1] See case recorded by Agnes F. Blackadder, M.B., the *B. M. J.*, 1901, vol. i., p. 765.
[2] "Clinical Medicine," p. 28, Griffin & Co., London, 1894.

Etiology. Tuberculosis of the mesenteric glands (tabes mesenterica) may occur at almost any age. I have met with it at 12 months and also at the age of 45, but it is relatively rare under 2 years of age, and distinctly rare at other ages. Male children appear to be more prone than females. The introduction of the tubercle bacillus is the proximate cause, and it has been generally supposed that this is introduced by the ingestion of milk from tuberculous cows, or milk which has otherwise become contaminated. If the mucous membrane of the alimentary canal is healthy there seems to be less risk of contamination. There is often a history of recurrent attacks of intestinal catarrh which have led to a weakening of the mucous membrane and the consequent entrance of the bacilli, but since more attention has been paid to the supervision of dairies and the sterilisation of milk the disease has undoubtedly become less frequent, and it seems probable that it will ultimately be stamped out. That so-called bovine tuberculosis may produce this disease is now proven.[1]

Treatment. Prophylactic measures consist in sterilising or asepticising the milk and regulating the supply whence it is obtained. In the remedial treatment the diet needs careful attention, both in its quantity and the quality. All food should be raised to the boiling point before administration. Small doses of creosote by the mouth or by enema, together with tonics, cod-liver oil, iodide of iron, and other remedies mentioned under phthisis (§ 98) are employed. Among local measures mercurial or iodoform ointment may be rubbed into the abdomen. When the peritoneum is extensively involved surgical measures are now employed very successfully, and some cases in which simple laparotomy has been performed even without washing out have resulted in recovery.

GROUP III. DEBILITY ONLY (ASTHENIA).

The causes of debility not necessarily accompanied either by pallor or emaciation are as numerous as those of the two preceding groups, and it must be remembered that all the disorders in both of those groups may commence with weakness only; in short, the majority of chronic disorders begin with debility. The fallacies (§ 391) and methods of examination have already been given.

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[1] Dr. Nathan Raw, *Brit. Med. Journal*, August, 1903, p. 470.

Commoner causes.

I. Senile decay and arterial disease.
II. Chronic interstitial nephritis.
III. Neurasthenia, or other incipient or obscure diseases of the nervous system.
IV. Chronic dyspepsia and obscure diseases within the abdomen.
V. Cardiac, tuberculous, and other obscure diseases within the chest.
VI. Diabetes mellitus and diabetes insipidus.
VII. Conditions referred to in Groups I. and II., in which anæmia or emaciation are ill-marked.

Rarer causes.

I. Myxœdema.
II. Addison's disease.
III. Bronzed diabetes.
IV. Disease of the pancreas, acromegaly, beri-beri, pellagra, and many other conditions mentioned in Groups I. and II.

When a patient is suffering from debility or loss of vigour of mind and body, without any very marked pallor or obvious loss of flesh, and without any marked physical signs or other evidences of disease, in the *first half* of life one would suspect neurasthenia; chronic dyspepsia or gastro-intestinal disorders; incipient or latent tuberculosis; diabetes.

In the *second half* of life one would suspect senile decay; chronic interstitial nephritis; obscure cardiac valvular or aortic disease; diabetes; myxœdema; Addison's disease.

And failing these, some of the conditions previously mentioned among the anæmic or wasting disorders (Groups I. and II.).

§ **419. I. Senile Decay** and **Arterial Disease.** As we advance in years the power both of body and mind notably declines. This should not be very obvious under 60, but the age at which it appears differs considerably in different persons, and still more in different families, for the onset of decay in persons, as in plants and animals, is largely a question of heredity plus the previous habits of the individual. Structurally there is a universal tendency to atrophy or degeneration of the parenchyma or functionally active tissues, and slight increase in the lower forms of tissue (such as the fibrous and supporting tissues) in all the organs and structures of the body. This is particularly seen in the cardiovascular system where it is the muscular coat, *i.e.*, the functionally active tissue of the arteries which first shows signs of senile degeneration.[1]

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[1] "On Senile Decay," Trans. Med. Soc. of Lond., 1897; and "On Arterial Hypermyotrophy and Medial Sclerosis," Trans. Path. Soc. of Lond., 1904.

Symptoms. Consequent on the changes just mentioned there is a universal lowering of vitality and nutrition, and the general enfeeblement of thought, word, and act which results in the mumbling, fumbling, and stumbling of old age. Physical weakness comes on so slowly that even the patient himself is hardly aware of it, and it is not sufficiently recognised that widespread disease of the arteries alone may give rise to progressive mental and bodily enfeeblement at whatever age it comes on.

The following case may be quoted by way of illustration:—Jessie T—— was admitted into the Paddington Infirmary in 1889 at the age of 49. At the age of 45 she began to complain of muscular and mental weakness. This gradually increased, so that at the time of admission she could only walk by pushing a chair before her, and the case was thought, therefore, to be some kind of paraplegia. There were absolutely no physical signs in any organ and no evidences of disease in the nervous system at any time, and the urine was always normal. She became progressively more and more enfeebled in body and mind, gradually took to bed, and died 10 years after admission of progressive asthenia. I was present at the autopsy in 1899, and all the organs were normal, both macro- and microscopically, with the exception of atrophy; but there was extreme and widespread disease of all the arteries of the body and of the brain, the main change being granular degeneration of the muscular coat of the heart and arteries, with consequent yielding and great dilatation of the arteries. In Infirmary work I have met with arterial disease in persons as young as 38 and 40—cases which were examined post-mortem—and weakness was the only symptom.

The condition of the heart and aorta should be carefully noted, especially any rigidity of the latter, as shown by accentuation of the second sound at the base and the character of a pulse-tracing (Fig. 33, p. 145). The arterial tension should be noted from day to day, and the walls of the superficial arteries carefully investigated (§ 67 *et seq.*). Among the later symptoms associated with senile decay of the cardio-vascular and other tissues, perhaps vertigo is the commonest. A large number of other vague cerebral sensations may be experienced, and even convulsions (senile epilepsy) may occur. The urine should always be carefully and repeatedly examined so that senile decay may not be confused with other causes of debility (*infra*), particularly chronic interstitial nephritis.

The *prognosis* depends a good deal upon the amenability of the cardio-vascular system to treatment. The diseases to which old age is most liable are of a chronic and degenerative nature, the arterial, *i.e.*, the nutritive system being responsible for this, and itself showing the most definite and widespread signs of degeneration. The immediate cause of death in old age is usually some pulmonary complication. An analysis of 409 fatal cases in persons of 60 years of age and upwards, who died consecutively in Paddington Infirmary—Feb. 1st, 1886, to Dec. 31st, 1892—showed that

121, or 30 per cent., died of some pulmonary condition (pneumonia, bronchitis, hypostatic congestion, and pulmonary apoplexy). The next most fatal disease was cancer, 62 cases (15·5 per cent.), then simple senile decay, 35 cases (9 per cent.), then contracted granular kidney, 24 cases (6 per cent.), then pulmonary tuberculosis, 22 cases (5·5per cent.).

The *treatment* should be mainly directed to the cardio-vascular system, and especially to the raising of low tension (p. 139) and the lowering of high arterial tension (p. 137).

Stimulants are nearly always called for in the treatment of disease in the aged. The food should be light, nutritious, and easily assimilable, and small in quantity: it is wonderful how small a quantity of food the aged require, and it has been reckoned that 12 ounces of solid food per diem are sufficient. It is not only useless but harmful to over-feed the aged; keep them warm and prevent chill, but do not over-feed them. Strychnine is *par excellence* the tonic of the aged.

II. **Chronic Interstitial Nephritis** (§ 298) (syn. chronic Bright's disease, granular or gouty kidney), should always be remembered as a cause of progressive enfeeblement coming on at or past middle life. It is indeed very apt to be mistaken for senility, and failing vigour is the leading symptom for which the patient seeks advice in a large proportion of both these conditions. Sometimes this weakness is accompanied by generalised muscular wasting, but quite as often there is none. The complexion is generally sallow, but there is no definite pallor till late in the disease. Headache is common, chronic interstitial nephritis being one of the commonest causes of headache coming on after middle life.

III. **Neurasthenia** (§ 518) and various other **functional** and **degenerative conditions of the nervous system** may be evidenced by general weakness. This is particularly the case in the functional disorders, such as neurasthenia and hysteria, where the weakness may amount to complete prostration. Such cases are usually met with in the first half of life or middle age. Among the gross lesions which are apt to come on insidiously with weakness are *paralysis agitans*, *bulbar paralysis*, and frontal *cerebral tumour*—diseases more often met with in the second half of life.

Myasthenia gravis is a rare condition coming on usually with generalised weakness. See also generalised paralyses, § 568.

IV. **Chronic Dyspepsia,** gastric dilatation, and other obscure **diseases within the abdomen** may be attended for a long time only with debility. Gastro-intestinal troubles produce a chronic toxæmia and mal-assimilation of food in which debility is a prominent symptom. *Mucous colitis* may be specially mentioned in this connection, also *appendicitis*, *abdominal cancer*, and many of the other conditions mentioned in Chapter IX.

V. **Obscure Diseases within the Chest** may be manifested by general weakness. *Incipient tuberculosis* of the lungs is a condition—occurring for the most part in younger subjects—which should always be remembered in cases of unexplained general debility. In the second half of life and especially in those with an alcoholic history, *cardiac enfeeblement* and fatty heart (§ 55) may frequently be unaccompanied by any other symptom than debility, and *aneurysm* of the third part of the aorta is another condition.

VI. **Diabetes mellitus** and **Diabetes insipidus** are often first revealed by progressive weakness, though our attention may also be drawn to these conditions by the thirst, polyuria, emaciation, or glycosuria (§§ 310 and 311). These diseases may occur in either the first or the second half of life.

In regard to the treatment of diabetes mellitus modern observers have shown that the dietary may be considerably relaxed under supervision by first putting the patient on a rigid dietary and afterwards adding various articles which are found not to increase the excretion of sugar. Particulars are given by Prof. R. Saundby in *The Lancet*, May 19th, 1900, and by Dr. R. Hutchison in *The Practitioner*, June, 1904. A strict milk diet has removed glycosuria even in young persons.

The **rarer causes of debility** not necessarily accompanied by pallor or emaciation are Myxœdema, Addison's disease, Diseases of the pancreas, Acromegaly, and some of the conditions mentioned in Groups I. (anæmia), and II. (emaciation).

§ **420.** I. **Myxœdema** (*μυζα*, mucous; *οιδημα*, swelling) is an insidious disease evidenced by weakness, lethargy, and other manifestations of deficiency in the metabolic processes of the body due to diminished thyroid function. It was so named by my respected teacher. the late Dr. W. M. Ord. on account of the mucoid degeneration which takes place in the subcutaneous tissue throughout the body, and is the most obvious anatomical change, producing a kind of solid œdema. It was at first believed by him to constitute a new and hitherto undescribed form of generalised œdema, but it is now known—and this was admitted by him—to be in reality a sporadic cretinism (§ 140) occurring in adult life. It is due to a diminished function of the thyroid gland, generally accompanied by a diminution in volume of the organ (see p. 267). It is not a very uncommon disease, but is frequently overlooked.

Symptoms. The weakness here takes the form of a very characteristic slowness of action, of thought, and of speech. It comes on very gradually and the patient frequently seeks advice for some other reason. (2) The aspect (Fig. 1, p. 23), however, is so characteristic that when the doctor has once seen a case he recognises it again directly. The face is slightly puffy and, the lines of expression being obliterated, it appears immobile and vacant; the puffiness of the eyelids may be mistaken for dropsy; the malar capillaries are injected and cause a characteristic flush on each cheek. The hair of the scalp and eyebrows is coarse, scanty and brittle. The speech is slow and drawling, the hands are flat, puffy and spade-like, and the nails brittle. All the movements are slow and the mental processes lethargic. (3) As the disease advances the skin of the whole body is thickened and at first sight gives the appearance of generalised dropsy, but myxœdema may be distinguished from the latter by the absence of pitting; puffy swellings may be noticed above the clavicles. (4) The urine is copious, of low specific gravity, and deficient in urea, but there is no albumin and no other change. (5) The thyroid is usually atrophied, though it is difficult to make this out, partly on account of the myxœdematous condition of the neck.

Diagnosis. The disorder may be mistaken in its earlier stages for *anæmia* and the other disorders mentioned in Group I., also from the other causes of *debility* now being considered; but the facial appearance is very characteristic. Later on it may be diagnosed from chronic interstitial nephritis and other forms of chronic renal disease by the absence of pitting on pressure and the absence of the urinary changes of renal disease.

Prognosis. Before the introduction of the thyroid treatment patients rarely lived more than a few years, dying usually of some intercurrent malady or complication. Mental changes, such as hallucinations and even dementia may supervene. Menorrhagia and other hæmorrhages are sometimes associated with the disease.

Etiology. The disease is much more frequent in women, in whom it supervenes usually about middle life. It is undoubtedly due to a deficiency of thyroidal function (athyroidism, p. 268), but what constitutes the proximate cause of thyroidal atrophy is not known. It is certainly not confined to any particular district as is endemic cretinism, and it differs from the latter in the age and sex which it chiefly affects.

Treatment. The treatment by the internal administration of the thyroid gland is so certain and efficacious that this may be used as a means of diagnosis. Tabloids of the extract, 4 or 5 grains, or one-third of a fresh sheep's gland may be administered twice or thrice daily after meals, gradually increasing until tolerance is established. This tolerance is known by the supervention of headache, giddiness, palpitation, or a general feeling of discomfort. The addition of arsenic to the treatment will sometimes relieve these symptoms and enable us to push the thyroid for a long time. Complete recovery may ensue after a few weeks, or few months' treatment. Relapses are very apt to occur upon ceasing the thyroid administration and the patient may be obliged to continue treatment indefinitely.

§ **421.** II. **Addison's Disease** is a rare malady, described by Dr. Addison[1] in 1851, characterised by progressive loss of strength, and general pigmentation of the skin, due to disease of the suprarenal capsules. The most common morbid change in the latter undoubtedly is tuberculosis, which may go on to caseation, fibroid, or other changes. Occasionally the suprarenals are affected in this disorder by malignant and other disease.

The *symptoms* come under five categories:—(1) Progressive *general weakness* is its most marked feature, and may appear long before any other symptom. It is unaccompanied, as a rule, either by anæmia, or marked emaciation, until perhaps towards the end. Uncomplicated cases present a sub-normal temperature throughout. (2) *Pigmentation* of the skin of a more or less general distribution ensues sooner or later. The colour begins with a yellowish tint, which gradually deepens into a bronze mahogany colour. The localities most affected are the exposed parts, the face, neck, and hands, and those where pigmentation is normally present, such as the axillæ, and nipples. The edge of a patch of colour shades gradually into the healthy skin around, which makes it difficult to discover such a patch in its early stage. The mucous membranes of the tongue, mouth and throat frequently present the same kind of patches. (3) *Gastric* symptoms generally occur at some time, such as vomiting, hiccough and cramp-like pains in the abdomen and loins. Pains in the limbs may also be complained of. The bowels are often constipated, but sometimes there is intractable diarrhœa which may be fatal. (4) *Cardio-vascular symptoms* may be present—palpitation, soft rapid and feeble pulse, dypsnœa, sighing, yawning, and later on a tendency to collapse. (5) *Nervous symptoms* are less common, but may consist of headache, vertigo, and nervousness. The mind is clear, excepting towards the end, when delirium, convulsions, or coma may set in. These five groups of symptoms vary in their predominance, but asthenia is always present, and pigmentation nearly always. There are two varieties, acute and chronic.

The *diagnosis* is often very difficult on account of the extreme vagueness of the symptoms, the absence of physical signs, and the resemblance of the pigmentation to various other cachectic states, especially cancer. *Cancer of the pylorus* is accompanied by sallowness, which is often mistaken for the pigmentation of Addison's disease. Both, moreover, are accompanied by enfeeblement, gastric pain, and vomiting. The diagnosis from *other pigmentary conditions* is given among the causes of pigmentation (Chapter XVIII.). Slight jaundice, the pigmentation of malaria, chloasma, and arsenical pigmentation may be borne in mind. *Chronic Bright's disease*, neurasthenia, and other conditions attended by asthenia mentioned in this group, are apt to be mistaken for the disease.

Prognosis. The course of the disease is progressive, and usually prolonged; it may last one to ten years. There are frequent relapses, with intermissions of comparative health, but it always terminates in death. It may end suddenly with syncope, severe vomiting, and diarrhœa, convulsions, or coma, or it may terminate gradually by asthenia. The commonest complication is tuberculosis of the lungs, or elsewhere; and pulmonary tuberculosis is the commonest cause of death.

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[1] "On the Constitutional and Local Effects of Disease of the Suprarenal Capsules," London, 1855.

Etiology. Patients are mostly about middle life, and by far the larger number are males. The essential cause is a defect of internal secretion of the suprarenal capsules, owing to their destruction by tuberculosis.

Treatment. Suprarenal extract may be tried in tabloids containing 1 grain (equivalent to 15 of the gland) twice daily, and in gradually increasing doses. The pathology of the disease rests upon the loss of the internal secretion of the suprarenal bodies, and if the secretion can be made good the fatal issue should be averted; suprarenal extract and fresh gland however has not hitherto been successful in arresting the disease. The symptomatic treatment consists in rest, and supporting the strength by cod-liver oil, tonics, good nourishment, and hygienic living, on the same principles as other tuberculous affections. Cold and over-exertion should be avoided.

§ 422. III. **Bronzed Diabetes**, or what should more properly be called "pigmentary cirrhosis of the liver with glycosuria," is a rare condition which might be mistaken for Addison's disease. In certain cases of hypertrophic cirrhosis of the liver, there is a considerable amount of hæmo-chromatosis which may give rise not only to the symptoms characteristic of cirrhosis, but to definite pigmentation of the skin. Certain of these cases may also be associated with a more or less permanent glycosuria, possibly owing to concurrent cirrhosis of the pancreas. The glycosuria usually occurs as a late event in such cases.[1]

IV. **Disease of the Pancreas, acromegaly, beri-beri, pellagra** and **other conditions mentioned in Groups I.** and **II.** (*q.v.*), may come on with debility only, or the patient may seek relief for this symptom.

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[1] See also Dr. Alex. Bruce, *The Lancet*, 1898, vol. i., p. 1692; Prof. Wm. Osler, *Brit. Med. Journ.*, 1899, vol. ii., p. 1595; and Auschütz *Deut. Arch. für Klin. Med.*, vol. 62, p. 411.

CHAPTER XVII.

THE EXTREMITIES.

In the preceding pages we have seen on several occasions that so-called local diseases, such as pneumonia and endocarditis, have by scientific research been shown to be only local manifestations of a general microbic infection. This principle will here again be illustrated, for a gouty joint is only the local evidence of disordered metabolism, and acute rheumatism has now been shown to be microbic in origin. In conformity, however, with the scheme of this work whereby all diseases are approached from a symptomatic standpoint, certain diseases, the symptoms and physical signs of which are referable mainly or entirely to the upper or lower extremities, will now be considered.

PART A. SYMPTOMATOLOGY.

The CARDINAL SYMPTOM referable to the extremities is **pain** (or painful sensations of some kind), which may or may not be accompanied by some **physical change.**

§ **423. Pain in the Limbs** should be investigated, like pain in other situations, as to its *position*, *character*, *degree*, *constancy*, and *duration*. Its position may be localised to the skin, or to a joint or any other structure, or be generalised, as in sheer exhaustion ; its character may be sharp and shooting (as in tabes) or dull and heavy (as in vascular lesions), or like pins and needles (as in nerve and neuro-vascular lesions). Pain in the limbs may come on *acutely* or *insidiously*.

(a) *Acute pain in the limbs* coming on more or less SUDDENLY may herald influenza, enteric fever, malignant endocarditis, variola, scarlatina, or some other specific fever. In many cases of influenza this pain and slight pyrexia are the only symptoms. *Acute rheumatism* also comes on rapidly with pains referable to the muscles, bones or joints, and so does *dengue* (break-bone fever).

Trichinosis is attended by excruciating muscular pain in the second stage of the disease when the parasite begins to migrate. A sudden sharp pain in one spot in the limb is felt when *embolism* of an artery occurs; so also in thrombosis of a vein; in both cases pyrexia may be absent.

(b) Pains in the limbs coming on more or less *insidiously* may be due to:—(1) *Peripheral neuritis*, and therefore all its causes, *e.g.*, alcohol, diphtheria, or syphilis. Long before such a disease as neuritis was recognised by the profession "pains in the limbs" were known to arise from excessive indulgence in alcohol, and in dealing with alcoholic subjects this should be remembered. The same pains may occur in neurasthenia, and it is quite possible that the pathological condition in this disease, as in alcoholism, neuritis, and the acute specific fevers, may be a toxic condition of the blood. (2) Pains in the joints or muscles are characteristic of *chronic rheumatism* and *gout*; when the pain seems to be in the bones *syphilis* should be suspected. (3) Pain affecting the nerves has a shooting or darting characteristic of its own, as in neuralgia, sciatica, or tabes dorsalis. (4) Growing pains (so called) in children are often of a somewhat serious import as being the only tangible evidence in them of subacute rheumatism, which may nevertheless be sufficient to produce endocarditis with permanent damage unless the condition is recognised and rest in bed with salicylates prescribed. (5) The "numbness" or tingling of the hands and feet known as acroparaesthesia (a word which indicates perverted sensations in the extremities) is very characteristic; it may be indicative of some vaso-motor disorder such as erythromelalgia (§ 430), or the incipient stage of tabes, general paralysis or other organic disease. (6) Severe pain in the foot should lead us to suspect flat foot or metatarsalgia. Metatarsalgia is a neuralgia of the foot due to lateral displacement of the heads of the metatarsal bones which press upon the nerves, and may produce a corn (for which indeed the patient may seek advice).[1] (7) Various diseases of the bones (§ 445) may come on insidiously with nothing more definite than vague pain in the limb or limbs. This must be specially remembered in children in whom pains of gouty or alcoholic origin are rare. Various forms of inflammation, acute or chronic, may arise, and unless the bone be superficial there may be no surface indications at all. Some of them such as osteomyelitis are very serious, and require prompt recognition. (8) A muscular strain or rupture of some muscular fibres may leave a chronic pain and partial loss of function (unattended by any physical sign) which is often hard to cure. In one of my cases the pain lasted over 5 years. (9) *Local injury* or pressure may cause pain, such as injury from a crutch, or sleeping in a cramped position, or lymphatic glands or other tumours in the axilla, neck, or pelvis. Shooting pains down the arms, especially the left, occur in aneurysm of the aorta (see also causes of single nerve paralysis, § 564).

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[1] J. Jackson Clarke, *The Med. Press and Circular*, June 14, 1899.

PART B. PHYSICAL EXAMINATION.

The physical signs referable to the extremities mainly consist of some visible or tangible alteration in the skin and general contour of the limb, the joint, the muscles, the bones, or the vessels and nerves.

§ 424. **Inspection of the Limb** may reveal generalised redness or alteration of colour, œdema, varicose veins, or some other diffuse or localised swelling. Eruptions prone to affect the skin of the extremities are specially dealt with in Chapter XVIII.

Even without the skill of a palmist or the acumen of a Sherlock Holmes a great deal concerning the temperament, habits, and diseases of a patient may be learned by a careful **inspection of the hands.** For instance, the long, thin, dextrous fingers, perpetually on the move, will almost surely indicate a nervous temperament and imaginative disposition, just as the short, thick, almost clumsy fingers and hands of another will bespeak slowness, deliberation, and doggedness. The occupation of a patient may often be learned from a glance at the palms. Some people habitually have cold, damp, clammy hands, and these are generally the subjects either of the alcoholic habit or the rheumatic diathesis, occasionally some other condition causing a defective vaso-motor tone. The *nails* can also afford us some information. They are dusky in all conditions of impaired circulation, and pale in anæmia; compression on the tip of the nail should not completely empty the capillaries, as it does in anæmia. In aortic regurgitation compression of the nail tip reveals a capillary pulsation. A transverse ridge or groove in the nails indicates an arrest of growth, and may mark the date of an illness or any disturbance of nutrition, of even so slight a nature as seasickness.[1] It is useful to remember that the nail takes about 5 or 6 months to grow from root to tip. Various distortions of the nail occur in neuritis and injury. Clubbed fingers, *i.e.*, fingers with a bulbous end and great convexity of the nails (filbert-shaped nails), are characteristic of congenital cardiac disease or valvular disease in early life. Emphysema and chronic phthisis and any disease attended with profuse expectoration may be attended by the same deformity. *Glossy fingers* (fingers with smooth, thin skin) are the result of a neurotic dystrophy, and are associated with destructive and paralytic lesions of the nerve trunks: they also occur in sclerodermia. Strumous dactylitis is a thickening of one phalanx due to disease of the bone, with infiltration of the tissues of the fingers, resulting in a deformity known as the "champagne bottle finger." It is met with chiefly in tuberculous, and sometimes syphilitic, children. "Heberden's nodes," lipping and distortion of the phalangeal joints, are in reality rheumatoid arthritis of the fingers. Gouty nodules of urate of soda form white masses near the joints, just beneath the skin, and have an external resemblance to Heberden's nodes. The bone ends of the wrists are enlarged in rickets, syphilis and pulmonary osteoarthropathy. "Spade-shaped" hands (with thickened tissues) are suggestive of myxœdema, and large, flat, ungainly hands with osseous enlargement of acromegaly and pulmonary osteoarthropathy. The "claw hand" (*main en griffe*) occurs as the result of injury, or neuritis of the ulnar and median nerves: it is also seen in progressive muscular atrophy, syringomyelia and cervical pachymeningitis. Wrist-drop is very characteristic of lead palsy.

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[1] *Vide Illustrated Medical News*, about 1890.

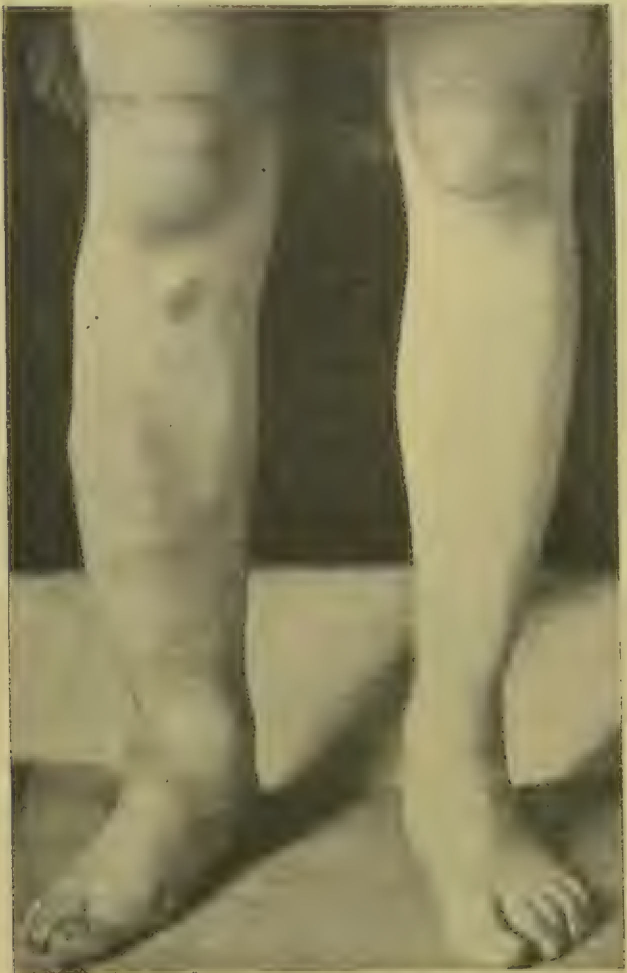

Fig. 120.—Elephantiasis telangiectodes, in a woman about 48 years of age.

Cyanosis (blueness) of the Extremities. Many people are born with a tendency to congestion, *i.e.*, blueness or redness, of the hands and feet. They appear to be the evidences of a vaso-motor instability or vascular want of tone. Such people are specially liable to chilblains and other kinds of erythema; in short, they might be called angio-neurotic subjects. The slighter degrees of cyanosis can be revealed by examining the nails and depressing the nail tip. The *causes of cyanosis* were fully discussed in § 27. Erythromelalgia, "dead hands," gangrene and Raynaud's disease are referred to below.

§ **425. Varicose Veins** consist of dilatation and tortuosity of the superficial veins, and are practically only met with in the legs, where their tortuous elevations produce obvious and characteristic alterations in the contour of the limb. They occur mostly in those who stand a great deal; they are more frequent in the female sex, and especially in those who have borne children. Varicosity of the veins predisposes to eczema and ulceration; and severe hæmorrhage may ensue from their rupture. The *treatment* of varicose veins belongs mainly to the surgeon, but it may be useful to mention here 4 hints which Mr. C. B. Keetley offers, viz. :— (i.) the more exercise the patient takes the better, especially in the form of bicycling; (ii.) the legs should be kept as cool as possible; (iii.) if the varicose veins occur on the thigh or the knee, operation is advisable in preference to the wearing of stockings or rubber bandages; (iv.) elastic stockings (the usual mode of treatment), should be made to lace. The best bandage however in my experience is the crêpe de voile; it is cool, light, and (if not ironed after washing) of very equable pressure.

Elephantiasis telangiectodes (Fig. 120) is a rare condition which is apt to be mistaken for varicose veins. It consists of a hyperplasia of the subcutaneous tissues together with a varicosity of the superficial veins which form loose masses like bunches of grapes beneath the skin. They are usually associated with a certain amount of superficial telangiectasis (dilatation of venules) in the skin over and around the masses.

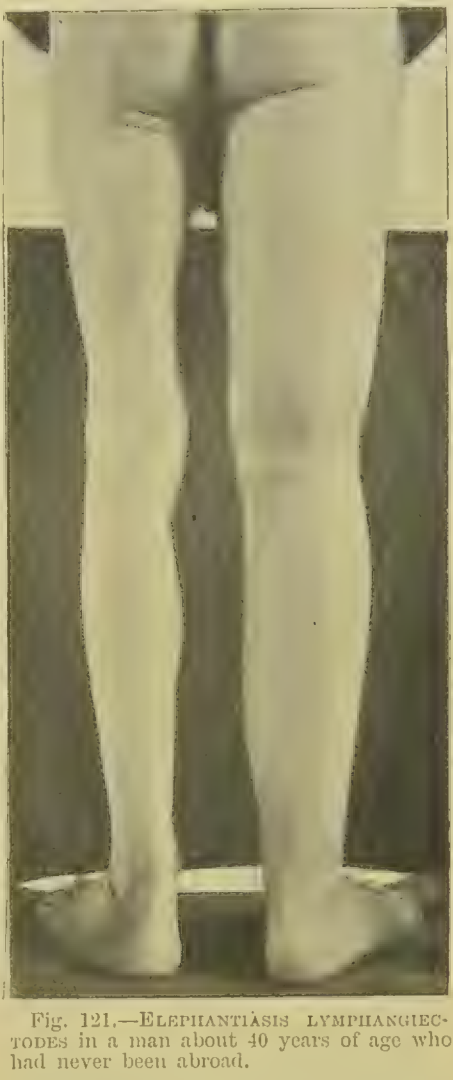

Fig. 121.—ELEPHANTIASIS LYMPHANGIECTODES in a man about 40 years of age who had never been abroad.

§ **426. Œdema of one Limb** (localised dropsy) produces generalised swelling which pits on pressure. The swelling due to the rare condition elephantiasis lymphangiectodes (see below) is of a much more solid character. Apart from *inflammatory œdema* and an extensive *angio-neurotic œdema* (§ 153), dropsy of one arm or one leg always points to some obstruction of the main vein of the limb by thrombosis within or pressure upon the vein. (1) *Thrombosis* (coagulation within the living vessel) with or without phlebitis (inflammation of the vein) is not uncommon in the femoral or iliac vein in the leg, and the brachial in the arm. In addition to œdema there are pain and tenderness at the seat of the obstruction, and a history or evidence at the time of some cause of thrombosis or phlebitis such as phthisis and other wasting disorders, any of the acute specific fevers, injury or local extension (as from an ulcer). The commonest example of thrombosis is *phlegmasia dolens* (or white leg) which is so apt to come on after confinement, partly as a result of the hyperinotic condition of the blood (*i.e.*, the excess of fibrin-forming constituents) which is associated with the puerperal state, and partly owing to previous pressure on the veins within the pelvis. Little can be done in the way of treatment beyond complete rest in the horizontal position, warmth applied to the limb and the administration of iron. Nothing will remove the obstruction, but in course of time the condition is relieved considerably, if not altogether, by the establishment of collateral circulation. (ii.) Œdema may also be due to *pressure upon a vein* by a tumour, such as enlarged glands in the axilla or elsewhere, aneurysm or other intra-thoracic growth pressing upon the veins coming from the arm; pelvic cellulitis, carcinoma of the uterus or bladder, bands of adhesion, hydatid, or other intra-pelvic growth pressing on the veins of the leg.

Elephantiasis lymphangiectodes (Fig. 121) is a solid œdema, not pitting on pressure in any notable degree, affecting one leg, occasionally one arm, or the scrotum, due to a blocking of the lymphatics of the limb. It is met with chiefly in tropical countries in persons whose blood at night contains the embryo of the filaria Bancrofti. The adult worm is believed to block the lymphatics and so produce the disease. It is, however, occasionally met with in temperate climates in persons whose blood does not reveal the parasite, and the cause of the blocking in these cases is obscure.

§ 427. **Swelling of the Lymphatic Glands** in the neck, axillæ, groins or elsewhere on the surface of the body or limbs may be due to:—1, injury and septic or infective processes; 2, tuberculous disease; 3, malignant disease; 4, syphilis; 5, acute specific fevers; 6, lymphatic leukæmia; 7, Hodgkin's disease; 8, lymphoma (?). The first three arise in glands adjacent to some focus of mischief and the glandular swelling usually remains localised; in the remainder all the lymphatic glands tend to become affected. Slight leucocytic changes occur in all, but particularly in lymphatic leukæmia.

1. *Local* injuries, septic sores, and abscesses give rise to enlargement of the neighbouring lymphatic glands. When a patient complains of pain and enlargement of the glands in the groin, for instance, these may be due to direct injury to or pressure on those glands; but one should always carefully inspect the foot for abraded skin around the toe nails, through which dirt or stocking dye may have been absorbed. *Post-mortem scratches* or inoculation from septicæmic cases are of a much more virulent nature. Red streaks along the course of the lymphatics indicate lymphangitis. The glands at the elbow and axilla become acutely painful and tender, and they may rapidly suppurate. This is a conservative process, for in this way the septic virus is prevented (usually) from extending to the general circulation. If, however, the virus is too intense or the dose be too large, general septicæmia and death in a day or two is the result before suppuration can occur.

2. *Tuberculous* disease of the lymphatic glands, especially of the neck, is very frequent in children. The disease is usually secondary to some other focus latent or active, but is generally localised to one group of

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glands and the process is slow and chronic. It is also recognised by the fact that the glands very soon become matted together into one solid mass, which in due time, if the case be untreated, undergoes caseation, breaks down and leaves a characteristic ulcer.

3. *Cancer* gives rise first to inflammatory enlargement of the adjacent glands ; later, the adjacent and distant glands become the seat of secondary cancer (§ 416). Lymphosarcoma is a sarcomatous growth *starting in* the lymphatic glands. It rapidly invades the surrounding structures, and the neighbouring glands.

4. Constitutional *syphilis* first affects the lymphatic glands in the neighbourhood of the chancre. They are small, hard (shotty), painless, and only perceptible on palpation, but for many years afterwards all the glands of the body, especially those in the groin, may be discovered on careful palpation to be thus indurated. They never suppurate with Hunterian syphilis as they do with a soft chancre.

5. In most of the *acute specific fevers* there is, as in syphilis, a slight generalised glandular enlargement. In those fevers which have a local manifestation, the throat in scarlatina and diphtheria for instance, the adjacent glands are first and chiefly affected. In bubonic plague the enlargement is very great. In certain milder cases of *plague* nothing but slight glandular swelling and a little fever occurs (pestis minor). Such cases are often overlooked, but they may give rise to epidemics of pestis major, or true plague. *Arthritis deformans* in children is accompanied by enlargement of the glands and spleen.

6. In *lymphatic leukæmia* there is a generalised glandular enlargement, but it is a subordinate feature to the enlargement of the spleen. The blood changes are very characteristic (§ 408).

7. *Hodgkin's Disease* (lymphadenoma, anæmia lymphatica) starts with a swelling of one group of glands which enlarges paroxysmally, generally attended with corresponding paroxysms of fever. The individual glands remain separate, painless, and may feel like a bunch of grapes. Each gland may attain a large size. Sooner or later other groups of glands become similarly involved ; but the spleen is never much increased (§ 409).

8. *Lymphoma* is a term applied to the enlargement of a single group of glands where no extension or constitutional symptoms occur. It is possible that lymphoma is a localised form of Hodgkin's disease.

§ 428. The JOINTS, MUSCLES, BONES, VESSELS, NERVES, and CONSTITUTIONAL SYMPTOMS should be next investigated.

The **joints** may need investigation for tenderness, pain, heat, swelling, or redness, and for loss of function or range of movement. The affected and the unaffected sides should be carefully compared. Slight degrees of fluid in a joint are often difficult to detect. The active movements (those which the patient can make) and the passive movements (those made by the doctor) should, with due consideration and caution, be tested. Among the *fallacies*, paralysis or muscular weakness is often simulated by chronic joint diseases and *vice versâ*, and pain in the limbs from various causes will often simulate a stiffness of the joint. In acute joint disease the fallacies of epiphysitis and acute osteomyelitis must be avoided.

The **muscles** may be investigated for tenderness, stiffness, or swelling. The investigation of paralysis, tonic or clonic spasm, or wasting, is given under diseases of the nervous system (Chapter XIX.). We are here concerned only with pain, tenderness, or swelling localised in the muscles; it is the presence of these localised symptoms which helps us to differentiate muscular diseases from paralysis and other diseases of the nervous system. To decide that the lesion is not in the bones or ligaments may be difficult; if it be in the muscle the pain is greater during active than passive movement of the affected muscle; if in the ligaments or joints the pain is about equal.

The examination of the **bones** belongs specially to the surgeon, but disease situated in the bones may be evidenced by pain, tenderness, swelling, or deformity. They often first come under the notice of the physician when pain is their only symptom, and diagnosis presents considerable difficulty.

In the diagnosis of SWELLINGS CONNECTED WITH BONES it is well to remember the following data.[1] Symptoms come on *acutely* with trauma, periostitis, osteomyelitis and deep abscess; slowly and *chronically* with caries, necrosis, chronic periostitis and osteitis, rickets, syphilis and tumour. In regard to physical signs the *diaphysis* is mainly affected in acute and chronic inflammation, in sarcomatous and other tumours; the *epiphysis* in rickets, syphilis and central sarcoma. The consistency of the swelling is *soft* in abscess and vascular sarcomata, *hard* in chronic inflammation. As regards the mode and rate of growth, the swelling *progressively enlarges* in inflammatory and malignant tumours, and is *stationary* in chronic inflammation and benign tumours; *receding* swellings are always inflammatory.

The **vessels** and **nerves** need examination when any of the symptoms indicate their implication, as in erythromelalgia and some other conditions in Group I. below. Pressure along their course may elicit tenderness, indicative of inflammation. The symptoms and effects of peripheral neuritis are given in Chapter XIX., and embolism of an artery or thrombosis of a vein in § 431.

The **viscera** should be examined, particularly in acute joint diseases, which are almost always the product of some blood disorder, *e.g.*, the heart must always be examined in rheumatic conditions, the kidney in gouty disorders.

Pyrexia and **Constitutional Symptoms** are present in a considerable number of diseases of the extremities, particularly in the acute joint and bone disorders; and they may be investigated on the lines laid down in Chapter XV. Rigors and sweating indicate a pyogenic process. Characteristic blood changes are found in several diseases, notably glandular, pyogenic and other septic processes.

PART C. DIAGNOSIS, PROGNOSIS, AND TREATMENT OF DISEASES CAUSING SYMPTOMS REFERABLE TO THE EXTREMITIES.

§ 429. Routine Examination and Classification. As a matter of routine, as in other cases, investigate—

First, the LEADING SYMPTOM, which in this instance is very often as visible or palpable to the patient as to the physician.

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[1] See also Mr. A. Pearce Gould's "Elements of Surgical Diagnosis."

Secondly, the HISTORY of the case, its mode of onset (acute or chronic), and evolution in chronological order.

Thirdly, examine the AFFECTED LIMB or limbs, their colour and contour, the joints, muscles, bones, vessels, or nerves, as may be indicated; and, finally, examine the VISCERA and the TEMPERATURE.

If there is any visible abnormality in the COLOUR or CONTOUR of the hands or limbs . . turn to § 424 *et seq.* or § 430
If the symptoms point to JOINT disease, acute or chronic . . . . . turn to § 434 or § 437
If the symptoms point to disease of the MUSCLES . turn to § 442
If the symptoms point to disease of the BONES „ § 445

GROUP I. ALTERATIONS IN COLOUR OR CONTOUR OF THE EXTREMITIES.

This group comprises only the following morbid conditions which can be considered to be medical. Alterations in contour, œdema of one limb, varicose veins, and enlarged lymphatic glands have already been referred to on pp. 775–779; they come mainly within the province of the surgeon. There remain

1. Erythromelalgia.
2. Gangrene.
3. Raynaud's disease.
4. Dead hands.

§ 430. **Erythromelalgia** (a term first used by Weir Mitchell[1]) is a painful redness and swelling occurring in paroxysms and symmetrically affecting both hands, sometimes the feet, and sometimes spreading to the arms and legs. One side may be more affected than the other, but I have not seen any cases in which both sides have not been involved to some extent.[2] The disorder starts intermittently with tingling and numbness in the extremities (acroparæsthesia), and later on a painful redness supervenes. The paroxysms are often determined and always aggravated by hanging the limbs down, and also by placing them in very hot or very cold water. They are often worse when the patient lies down and goes to sleep, and thus the night may be sadly disturbed. The pain and swelling are lessened by holding the hands over the head. There is no paralysis, but owing to the numbness and swelling the fingers cannot easily be bent. The swelling and redness affect the whole hand (Figs. 122 and 123)—not patches as in chilblains, lupus, or erythema. On this account the *diagnosis* is not difficult; in the cyanotic form of *Raynaud's disease* the symptoms start and prevail in one or two finger tips. In erythromelalgia all the fingers and the whole hand are about equally involved. Thickening of the subcutaneous tissues may ensue and the paroxysms are apt to return even after long intervals. It is a prolonged and very painful disorder, but it is not fatal, and is in my experience to some extent amenable to treatment.

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[1] *Philadelphia Med. Times*, Nov. 23, 1872.
[2] See a clinical lecture on this subject in *The Lancet*, June 1, 1902.

Etiology. The female sex is far more prone to the disease; out of 37 consecutive cases which I observed between 1900 and 1902 only 2 were males. It appears to arise more frequently between the ages of 18 and 25, and at the climacteric. The rheumatic, gouty, and hysterical diatheses predispose to the complaint; and the determining cause is, in my belief, some altered condition of the blood. Several cases which I have observed have exhibited, concurrently with a severe paroxysm of the erythromelalgic symptoms, erythematous blotches on other parts of the body, and severe "rheumatic" pains in the limbs. The disease must, I think, be regarded as a vaso-motor paralysis due to a toxæmia; associated probably with an inherent instability of the vaso-motor centres controlling the ends of the extremities.

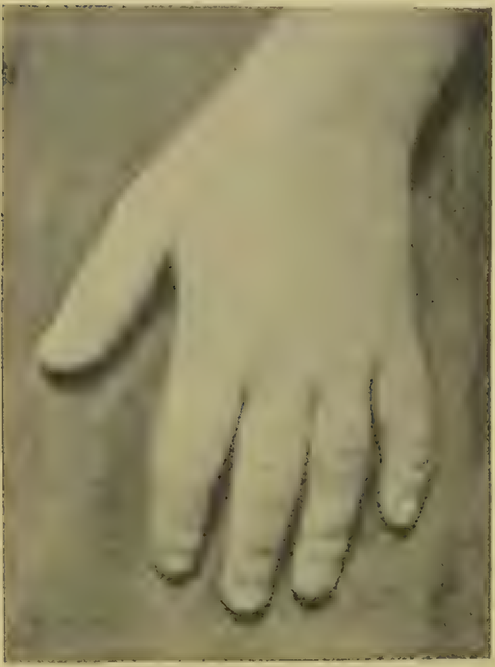

Fig. 122.—Erythromelalgia in a woman aged about 30.

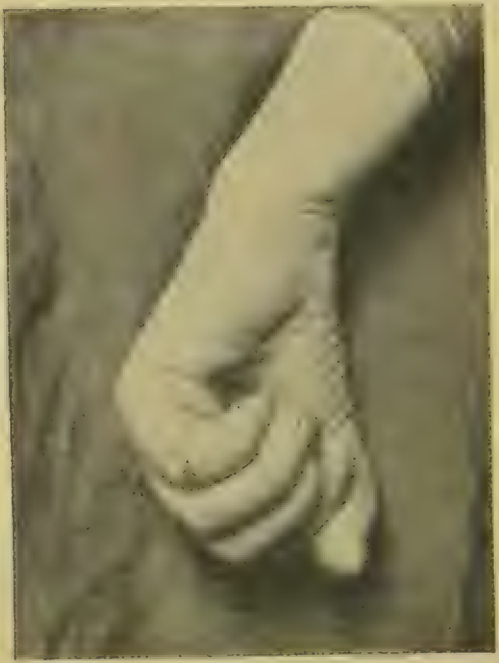

Fig. 123.—Erythromelalgia, showing maximal closure of the hand.

Treatment. Bromides invariably relieve the condition for a time; arsenic, strychnine, quinine, and other tonics are useful. Ergot sometimes does good. The general health should be attended to and particularly the digestion; bicarbonate of soda being especially beneficial. I have tried salicylates and anti-rheumatic treatment, but not with very marked success. A weak descending galvanic current is the most efficient curative agent in my experience; the positive pole is placed on the nucha, a divided negative pole at the bottom of 2 jugs of water in which the patient's hands are immersed for 10 to 15 minutes once or twice a day.

§ 431. **Of gangrene,** necrosis or death of part of an extremity, there are two kinds—

(a) In DRY GANGRENE the extremity becomes white and cold, then of an ashy and black colour; the part shrivels up, becomes dry and mummified. It is chiefly met with among old people—senile gangrene—and is due to the gradual obliteration of the lumen of the artery supplying the part, combined with more or less cardiac enfeeblement. It is also met with in younger patients in Raynaud's disease, and in cases of embolic blocking of an artery. The artery is tender at the seat of the embolism, and ceases to pulsate below.

(b) In MOIST GANGRENE the part becomes cold, purple or mottled, and engorged with blood. Blebs then form on the surface and a bright red line separates the dead from the living tissues. The dead part ultimately sloughs off and leaves an ulcer. This gangrene is due to venous obstruction, the result of thrombosis, pressure, injury or inflammation. The gangrene occurring in diabetes is of the moist variety. The treatment of both of these conditions belongs to surgery, but warmth, the posture of the limb, and the administration of diffusible stimulants are points to be attended to.

§ 432. **Raynaud's Disease** (syn. Symmetrical Gangrene; Local Asphyxia of the extremities). This disease, which was first described in 1862 by Dr. Raynaud,[1] is characterised by local vascular changes in one or more of the fingers, for the most part symmetrically on the two sides of the body, resulting very often in gangrene. Three types or stages of the disease have been described—a syncopal type, due to vascular spasm, an asphyxial type, due to vascular dilatation, and a gangrenous type.

Symptoms. Usually, the first thing noticed is a pallor (*local syncope*) and numbness of one or more of the fingers or toes, usually the corresponding finger or toe on both sides, coming on in attacks, lasting an hour or more. This pale or syncopal stage is generally followed by a reactionary stage of congestion and heat with swelling and lividity (local asphyxia), in which the tip of one or more of the fingers or toes, or the ears, may be of a dark purple hue. There is usually a good deal of pain. Sometimes the pale stage is very marked, sometimes it is wanting or it may be so transient as to be unobserved. Occasionally the entire hands are involved. After a certain number of these attacks *gangrene* occurs at the tip of one or more fingers or toes, the dead becomes separated from the living part in the usual way, and the ulcer that is left heals normally, but slowly. Cases have been recorded[2] of extensive multiple gangrene in which the patient has lost entire limbs in this way. The attacks just described may be the only symptom, but in the majority of cases other symptoms of considerable pathological interest may be observed. In a certain number of cases there is a generalised scleroderma, the skin having the appearance of being stretched, and smooth or sometimes cracked; and in such cases all the fingers are pale and dead-looking, and their entire substance becomes wasted. In a certain number of cases erythematous blotches occur from time to time in different parts of the body, which leave bruise-like stains. The patients are usually highly nervous, and prone to emotional attacks. Transient attacks of hemiplegia and aphasia have been observed, and attacks of paroxysmal hæmoglobinuria, all pointing to vaso-motor irregularities in other parts of the body. Effusion into the phalangeal and other joints may supervene and may result in ankylosis.

The *diagnosis* is not usually difficult. In the earlier stages it has considerable resemblance to erythromelalgia and to "dead hands," but these affections are not so localised to the finger ends, are less severe and never go on to gangrene.

Prognosis. The disease runs a prolonged course of many years with a series of attacks which become gradually more prolonged and frequent, and the patient gradually becomes more and more helpless. There are many degrees of severity of this disease, ranging from what amounts to no more than a small localised syncope or asphyxia to gangrene of the entire segment of a limb. It is a curious circumstance which I believe to be uniform that once a finger has become gangrenous the stump does not become similarly affected later on. The subjects of this malady in a marked form rarely reach old age, but usually die of some intercurrent malady.

Etiology. The disease is more common in women and especially those of a nervous diathesis. It appears for the first time usually between the ages of 15 and 30. Attacks may be brought on by chill or mental disturbances. The pathology of this strange disorder is unknown, but it is undoubtedly a vaso-motor affection, and in view of its symmetry is very probably a derangement of the local vaso-motor centres in the cord.

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[1] "Thèse de Paris," 1862, a thesis written for the M.D. at the Paris University in that year.

[2] Trans. Roy. Med. Chir. Soc., Lond., vol. xii.

Treatment. The affected limbs must be kept warm with cotton wool, and the patient protected from exposure to cold. The most efficacious remedy for the latter is undoubtedly electricity, as originally suggested by Sir Thomas Barlow. The method I have described under erythromelalgia may be employed, and as strong a current as possible used. Sir Thomas Barlow recommends the constant current, but one case under my care derived equal benefit from the interrupted current. Thyroid gland and nitroglycerine have been used and would certainly be indicated in the syncopal type. The pain is intense and may require morphia, which acts in a double way in asphyxial cases by giving tone to the vessels.

§ 433. **Dead Hands** (pallor of the hands). Many patients--but particularly those who present other evidences of an inherent vaso-motor instability, and are subjects of the gouty or rheumatic diathesis—complain that the hands or finger tips "go dead," or white, like those of a corpse, and feel numb and cold. These attacks, which rarely last very long, may happen in warm summer weather, without any obvious cause or consequent on anything which produces a nervous or emotional condition. This vascular disorder appears to be the converse of erythromelalgia, the fingers or hands being pale or shrunken instead of red and swollen. It is akin to acroparæsthesia and erythromelalgia, but it most resembles the slight or early phase of Raynaud's disease. The *treatment* should be directed to the diathesis upon which they depend, combined if necessary with muscular exercise and electricity. These attacks are not as a rule serious.

GROUP II. JOINT DISEASES.

The methods of examination and exclusion of fallacies have already been described. Arthritic disorders may conveniently be grouped into acute and chronic.

acute.

I. acute gout.
II. acute rheumatism.
III. acute gonorrhœal arthritis.
IV. pyæmia.
V. acute specific fevers.
VI. purpura, scurvy, hæmophilia.
VII. traumatism.
VIII. extension from adjacent bone.

chronic.

I. chronic gout.
II. chronic rheumatism.
III. rheumatoid arthritis.
IV. gonorrhœal arthritis.
V. chronic septic processes.
VI. tuberculous synovitis.
VII. syphilitic arthritis.
VIII. hysterical joint disorder.
IX. neuropathic joint disease.

a. *Acute Joint Diseases.*

Acute joint diseases, a list of which has just been given, come on more or less abruptly, and are as a rule attended by the local and general signs of inflammation. Acute rheumatism is essentially an erratic polyarthritis from the commencement; acute gout usually affects a single joint; most of the other causes start in one joint but (excepting VII. and VIII.) tend to a progressive involvement of others. It is worth noting that all the acute joint disorders (traumatism being excluded) are due either to some microbic process (II. to V.) or to some other blood disorder (I. and VI.). These facts emphasise the necessity of investigating the constitutional symptoms, the viscera, and the blood.

§ **434.** I. **Acute Gout.** Gout is a diseased or disordered metabolism associated with excess of uric acid in the blood, characterised by recurrent attacks of acute inflammation of the joints with deposition of sodium urate. It is one of the oldest known diseases. Gout occurs in acute, chronic, and irregular forms.

The *symptoms* of an attack, or paroxysm, of acute gout are usually heralded or preceded by gouty dyspepsia, heartburn, flatulence and weariness after food, fulness and tenderness in the epigastrium and liver, indentation of the tongue, a bad taste in the mouth, and excessive secretion from the fauces in the morning, scanty, high-coloured urine constantly depositing urates, cardiac irregularities and intermissions in the pulse, restlessness at night, and a tendency to catarrh of the mucous membranes on the slightest exposure. The onset of an attack is usually *very sudden*, often in the middle of the night. It affects preferably one of the smaller joints, and especially the big toe. The swelling in a marked case is tense, shining, red, pits on pressure, and is acutely tender, but suppuration never occurs. Other joints may become affected, but the inflammation does not shift from one joint to another as in acute rheumatism. Mild constitutional symptoms are present with pyrexia (102°); there may be muttering delirium at night. The urine contains less uric and phosphoric acid than before the attack, and may contain a trace of albumin. An attack lasts from 2 to 3 days or 2 to 3 weeks. After an attack the health is frequently better than it was previously; but the intervals between the attacks gradually become shorter as time goes on, at first 2 or 3 years, then 1 year, then 6 months; finally the disease becomes chronic, and permanent changes take place in the joint. *Chronic gout* and its associated symptoms are described under chronic joint diseases, § 437.

Varieties. (1) The symptoms of irregular or *atonic gout* consist chiefly of dyspepsia and a variety of symptoms referable to various organs of the body, supposed to be due to a deposit of gouty materials therein (see complications below). (2) *Retrocedent* or suppressed gout is a term applied to cases where the joint mischief suddenly improves coincident with internal symptoms affecting the digestive tract, the heart or the brain. Thus there may be vomiting and diarrhœa, dyspnœa, arrhythmia, even pericarditis, delirium and coma, or cerebral hæmorrhage. These symptoms are often associated with chronic interstitial nephritis, which is one of the consequences of gout.

The *diagnosis* of acute gout is not difficult excepting sometimes from acute rheumatism.

DIAGNOSIS BETWEEN ACUTE GOUT AND ACUTE RHEUMATISM.

ACUTE GOUT.	ACUTE RHEUMATISM.
In typical cases—	In typical cases—
Middle age; male sex.	Youth; either sex.
Preference for smaller joints; never wandering from joint to joint.	Preference for larger joints; usually wandering from joint to joint.
Swelling is usually red, tense, pitting on pressure, acutely tender.	Swelling is hot, but pale, tender only on movement of joint.
Ears show tophi.	No tophi.
Fever may be slight or transient.	Fever always marked and continuous.

Prognosis. The duration of an attack or paroxysm—which is rarely fatal in itself—depends mainly upon the age and constitutional condition of the patient. When, however, symptoms of suppressed gout come on the case may end fatally with great suddenness. Gout tends to shorten life mainly by the resulting kidney disease and cardio-vascular changes, and the ultimate prognosis largely depends upon the condition of the urine, which should be of good specific gravity and free from albumin. Among the complications and so-called *irregular* forms of gout, (1) chronic interstitial nephritis is the most important. During an attack there is generally a certain amount of albuminuria owing to congestion of the kidney, or deposit of urate of sodium, but this passes off. Gradually, however, after repeated attacks, an interstitial fibrosis takes place in the kidney, which from this association is known as the gouty kidney. Lithuria or lithæmia (§ 251) is by some considered an irregular form of gout. Renal calculus occurs in persons of the gouty diathesis who may have escaped joint symptoms. (2) Cardiac and cardio-vascular diseases come next in frequency, and consist of various valvular lesions and a thickening and degeneration of the arterial walls. (3) Bronchitis of a subacute or chronic form is frequent, and in treating this the gouty condition must not be forgotten. (4) Various gastric and hepatic derangements are frequent in gouty subjects, especially congestion of the liver. (5) Eczema and other skin diseases of an intractable kind supervene. Hot itchy eyeballs, migraine and episcleritis often are present.

Etiology. Among the *predisposing* causes of gout, age, sex and heredity are extremely important. (1) As regards age, the disease is rarely met with under 30, and the tendency increases up to the age of 50. It rarely starts for the first time over that age. (2) The disease is almost confined to men; if it occurs in women the attacks are generally very slight. (3) Out of 520 cases collected by Sir Alfred Garrod, 332, or nearly two-thirds, were distinctly hereditary. Like landed possessions the predisposition is transmitted mainly through the male line; but it sometimes skips a female generation and reappears in the sons. (4) Lead in the system is also a strong predisposing factor; painters, glaziers, etc., are very prone to gout and gouty kidney. (5) A fulness of habit, with feeble circulation, is a predisposing factor. (6) Attacks are more frequently met with in the changeable weather of the spring and autumn. The *exciting* causes are—(1) the use of alcohol, and especially those forms which contain a high percentage of both sugar and alcohol, such as port wine, brown sherry, Madeira, sweet wines generally, and malt liquor. (2) Nitrogenous food in excess is credited with being able to produce gout; at any rate it is commoner in butchers and meat eaters than among vegetarians (compare remarks below). (3) Both of the foregoing causes are more potent when combined with deficient exercise in the open. (4) It is a curious and imperfectly explained fact that gout is very rare in Scotland, at least among Scottish artisans. A possible explanation of this exemption is that the beverage of the Scottish artisan is whisky while that of the English workman is beer. An attack may be determined by (1) a debauch of alcohol, especially of certain kinds; (2) indigestion; (3) a chill to the surface of the body; (4) severe mental or bodily fatigue; (5) injury to a part, which will not only determine an attack but also the particular part affected. There is, however, a tendency for attacks to recur in the same joint.

The discussion of the pathology would be out of place here, but it may be repeated that the clinical phenomena are due to the presence of uric acid in abnormal amount. This was first demonstrated by Sir Alfred Garrod, who put 2 drachms of blood serum obtained by bleeding or blistering into a watch glass, added 12 minims of strong acetic acid and left a small thread in the fluid; in a day or two it became covered with uric acid crystals. Gout is due to disordered metabolism in some way connected with diminished alkalinity of and excess of uric acid in the blood. Sir William Roberts pointed out that in normal conditions uric acid circulates in the blood and is excreted in the urine as a quadriurate: in gout the quadriurates accumulate in the blood either from deficient elimination by the kidneys or over-production. By taking up an atom of the sodium carbonate in the plasma this quadriurate becomes converted into a biurate of sodium. The biurate, being less soluble than the quadriurate, becomes deposited in tissues which have low temperature, poor circulation, and a high percentage of sodium chloride, such as cartilage and fibrous tissue. Dr. Woods-Hutchinson[1] maintains that "the uric acid of gout, like the phosphoric acid which invariably accompanies it, is merely a result and measure of the destructive metabolism of the nucleins of the body cells, chiefly probably of the leucocytes, in response to the invasion of poisons or toxins, either organic or inorganic (lead, phosphorus, alcohol, acetone). . . . As most of the toxins . . . are of intestinal origin or entry, *diet in gout should be regulated solely with regard to the diminution of intestinal fermentation and putrefaction.*" Hence, as he shows, meat in itself is not a cause of gout, and in many gouty subjects does not cause gout except when taken together with carbohydrates which decompose in the digestive tract.

Treatment during an attack resolves itself into dietetic and medicinal measures. (1) It follows from what has just been said that a low diet of milk and farinaceous food, and complete abstinence from alcohol, should be enjoined unless the heart be fatty, when well diluted pure spirit is the only form permissible. (2) A brisk cathartic with one or more grains of calomel should be given at the onset, followed by frequent doses of saline purgatives, such as Hunyadi Janos water, or Carlsbad. (3) Alkaline carbonates (potassium, lithium, sodium) are very efficacious in promoting the solution of uric acid.[2] (4) Colchicum (combined with alkaline carbonate) used to be regarded as a specific, and it may be given every 4 hours (♏40 for first dose, then ♏12, or F. 87) until the pain is gone, then it should be stopped. (5) Opium may be needed for the pain, but should not be given if there be albuminuria, or suspected renal changes. (6) Local treatment consists of complete rest, wrapping the joint in cotton wool, and the application of sedatives to the joint, such as lotions of sod.

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[1] "The Meaning of Uric Acid and the Urates," by Dr. Woods-Hutchinson, *The Lancet*, Jan. 31, 1903, p. 288.

[2] Sir Wm. Roberts first showed that potassium rather than sodium salts should be given in acute gout. Dr. A. P. Luff (*The Lancet*, March 30, 1900) has shown that acute gout is due to the conversion of soluble gelatinous biurate into insoluble crystalline biurate. This process is hastened by sodium, and delayed by potassium salts. This explains the beneficial action of potassium salts noticed by the first observer.

bicarb. ʒiv., with laudanum ʒij. in ℥x. of water. I have also found a very comforting lotion consisting of:—sp. vin. rect. ʒiij.; liq. amm. acet. ʒiij.; aq. rosæ ʒiij.; aq. ad ℥xij. When symptoms of suppressed gout come on, employ eliminatory treatment promptly, stimulate if symptoms of collapse follow, and apply counter-irritation (mustard, turpentine stupes) and hot fomentations to the chest or abdomen as the case demands.

Treatment between the attacks, i.e., preventive treatment, resolves itself mainly into a question of diet, and the treatment of gouty dyspepsia. The dietetic treatment is of importance, as in chronic gout. The nitrogenous food-stuffs should be reduced because they throw too much work on the liver and kidneys; the carbohydrates because they consume more oxygen and are apt to ferment in the intestines; and the sugars and fats, particularly the cooked fats, because they tax the liver too much. Sweet wines, malt liquors, pastries and all sugar, sweet and greasy dishes must be forbidden; while plain food and abundance of pure water (to aid elimination) must be ordered. The hygienic treatment consists of regular exercise in the open air, and in aiding elimination. Among the remedies for the elimination of uric acid the most valuable in my experience is piperazine; it may be given five grains t.i.d., or in the form of an artificial mineral water. Lysidin, urotropin and uricedin have also been recommended. An occasional dose of mercury followed by a saline is useful. As regards drugs colchicum is of little use, except in the sub-acute exacerbations, when it may be given with large doses of pot. iod. until the pain is relieved. Guaiacum, given in the form of the resin, grs. 5—10 in cachets, is a valuable preventive of the gouty paroxysms. Locally blisters or iodine may be employed near the joints; or alkaline lotions (carb. of lith. or sod. gr. 10 to the oz.). Massage, hot air and radiant heat treatment is also useful (see chronic rheumatism). Mineral waters (Carlsbad, Vichy, Hunyadi Janos, Friedrichshall) should be freely used. Potassium or sodium bicarbonate has a beneficial effect upon the alimentary canal, and it is a very good plan to order a small teaspoonful in half a pint of warm water to be drunk every night and morning. Some advocate the use of potassium instead of sodium salts, and cases are reported which remained free of gouty symptoms when taking potassium chloride instead of common salt with meals. Visits to Bath, Harrogate, Buxton and Strathpeffer in this country are undoubtedly beneficial because of the regulation of the life enforced there. Carlsbad, Royat, Aix-les-Bains, and a number of other foreign spas, are annually visited. Gouty dyspepsia may be treated by the mineral waters just mentioned, or on the principles laid down in Chapter X. The best tonics are nux vomica, arsenic, and if necessary small doses of peptonate of iron.

§ 435. II. **Acute Rheumatism** (rheumatic fever) is an acute febrile disease due to a toxic condition of the blood, formerly believed to be lactic acid, but now shown to be microbic by Poynton and others, with erratic painful swellings of the joints and a marked tendency to disease of the heart; running a prolonged course of many weeks if untreated, and followed by a great tendency to relapse. The poison of rheumatic fever tends to affect not only the joints, but all the fibrous, serous and muscular tissues; but the joints, endocardium and pericardium (which, it will be observed, histologically resemble each other) are the favourite situations of the inflammation. Acute rheumatism, unlike acute gout, attacks several joints, usually the larger ones, *e.g.*, the knees, ankles, shoulders.

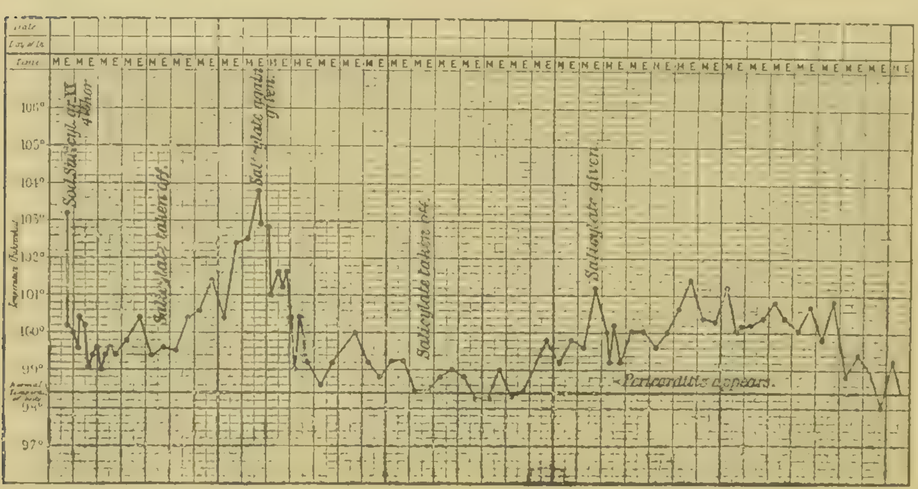

Fig. 124.—Rheumatic fever. Henry H——, æt. 22; the chart shows efficacy of salicylates in reducing the temperature until pericarditis appears; then the controlling power of the drug is less.

Symptoms. (1) The fever, which may have been preceded by tonsillitis for a day or two, comes on in the course of 24 hours, setting in before or at the same time as the joints are inflamed. It is of a continued type (Fig. 124), usually remaining about 102° or 103° F. for some days. The onset of any inflammatory complication in the pericardium or elsewhere is marked by an exacerbation of fever, and sometimes by pain, and delirium which is otherwise *extremely rare* in acute rheumatism; in uncomplicated cases the mind remains quite clear throughout. The usual accompaniments of pyrexia are present, viz., the urine is scanty, highly coloured, loaded with lithates, with an excess of urea and deficiency of the chlorides; the tongue is coated, the pulse quick and bounding, usually over 100. The blood exhibits leucocytosis. (2) In adults there is a profuse perspiration with a sour disagreeable odour and an acid reaction, but in children this is unusual; later on sudaminal vesicles are frequently seen. Erythematous and other rashes occasionally appear. (3) The two distinguishing features of the joint lesions of acute rheumatism are their wandering or metastatic character, and the absence of suppuration. The effusion into a joint is not very great; first one joint is affected, by the next day another is involved, the first joint having almost recovered; finally several may be affected together. The joints are hot and swollen, and though not tender to the touch, are acutely painful on the slightest movement. The skin over the joints is either unaltered in colour or shows a faint flush. 4. Peri- or endocarditis are so frequent as to be almost part of the disease; the pericardium may be the first serous membrane to be affected. 5. In untreated cases the fever and local inflammation may subside gradually in 4 to 5 weeks, and return again after an interval lasting perhaps a few days to a fortnight. In no other acute specific disease, excepting perhaps diphtheria, is the blood so deteriorated in so short a time; the patient shows grave anæmia during convalescence. Even after recovery the liability to recurrence is very great, and special care is needed.

Three *variations* of the above symptom-group are met with clinically. In *subacute rheumatism* all the symptoms are milder, and may drag on for months. *Malignant rheumatic fever* is a very serious form in which the heart is mainly involved, the joints little if at all. An eruption something like typhus may appear, and after a few days the temperature rapidly rises and the patient dies. *Rheumatic fever in infancy* is rare, and differs in 4 ways—(1) the local signs in the joints are not prominent; (2) the heart is more commonly affected (74 per cent.);[1] (3) nodules are frequent (16 per cent.); and (4) chorea is a common sequel. Its detection may be difficult as it may closely resemble infantile scurvy, which is known, however, by the swollen gums and failure of treatment by salicylates. Syphilitic epiphysitis and arthritis are known by the great local tenderness and œdema extending beyond the joints. Rheumatism in infancy may also be mistaken for infantile paralysis, and acute tuberculous hip-joint disease.

The *diagnosis* of rheumatic fever in the adult is not as a rule difficult. Acute gout is distinguished by its sudden onset, and by the other features mentioned in the table given above. Acute rheumatoid arthritis affects chiefly the small joints of the fingers (§ 439). Pyæmia (when arising from some internal cause) may closely resemble rheumatic fever, but in pyæmia the joint inflammation is not erratic, the pyrexia is typical, accompanied by rigors, cerebral symptoms, and an enlarged spleen. Among the other diseases which sometimes have to be diagnosed are—dengue, which has a characteristic eruption; trichinosis, in which the pain and swelling are referable rather to the muscles, and are preceded by gastric symptoms; ulcerative endocarditis, in which the joint swelling is absent, and the temperature intermittent; and osteomyelitis starting in the epiphysis, and spreading to a joint.

Prognosis. The disease is not dangerous to life when it attacks the joints only, but the complications are serious, and one attack predisposes to future attacks. The untoward symptoms are hyperpyrexia and cerebral symptoms. An attack is grave in proportion to the height of the temperature, the cardiac complications, and the presence of cerebral symptoms. The latter, happily rare, are of the gravest import unless accounted for by salicylates. The visceral *complications* of rheumatic fever are more serious than the disease itself. The chief of these relate to the heart, which should in all cases be examined daily as a matter of routine. Pericarditis is extremely common, and may supervene without pain, and so escape notice. In children it may become purulent. Simple endocarditis specially affecting the mitral valve[1] is equally common; ulcerative endocarditis is rare. Myocarditis and dilatation also occur. The liability to all of these complications increases with each attack. Pharyngitis and tonsillitis (in 80 per cent. of the cases according to some observers) are frequently present. Pleurisy is common, but pneumonia, bronchitis, erythema nodosum, purpura (peliosis rheumatica) and iritis are less common complications. Rheumatic nodules, small subcutaneous fibrous structures, may be found about the knees, elbows and spine, especially in children towards the end of the attack. Anæmia and chorea are frequent sequelæ; chorea may also be the first sign of rheumatism in a child.

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[1] Dr. G. F. Still, *Clin. Journ.*, vol. ii., p. 390, No. 22, Mar. 23, 1898.

Etiology. Age is the most important predisposing factor, acute rheumatism being almost confined to persons under 25, the commonest age being between 10 and 20. It is comparatively rare under 10, and extremely rare in advanced life. Males seem slightly more prone to the disease, and heredity plays a considerable part. Among the determining causes may be mentioned exposure to cold or chill and fatigue. Acute rheumatism is apt to follow an attack of scarlatina or chorea, just as these in turn may succeed an attack of rheumatism.

Rheumatic fever has for a long time been regarded as a blood disorder of *metabolic* and *chemical origin*, lactic acid or its combinations being believed to be the peccant material (Prout, Latham, and others).

Some have attempted to maintain that these metabolic changes were due to some *nervous defect* after the manner of neuropathic arthropathy. On the other hand an epidemic prevalence in rheumatic fever has long been noticed by careful clinical observers. Dr. Arthur Newsholme in 1895[2] maintained that rheumatic fever was a specific febrile disease, due to the introduction of a micro-organism from without. By a study of death— and hospital—statistics and of its seasonal and annual incidence he showed that the disease was epidemic, its maximum incidence being in January, its minimum in August: that it bore a direct relationship to a low level of the ground water; that it varied inversely as the rainfall; and finally that the humidity of the atmosphere had no appreciable effect. The clinical evidence on the resemblance of the symptoms of acute rheumatism to those of septic infection—especially in its involvement of the endocardium and the serous membranes—also supports the view of its being a specific infective disease. But it was not until 1900 that Drs. F. J. Poynton and Alexander Paine[3] demonstrated that the disease was due to a diplococcus, which after being isolated answered all the experimental tests of specificity (p. 572). These researches have now been abundantly confirmed.

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[1] In 150 fatal cases analysed by Dr. F. J. Poynton, mitral endocarditis existed in 149. Dilatation of the heart was also very common.

[2] The Milroy Lectures, *The Lancet*, 1895, vol. i., pp. 589 and 657.

[3] Dr. F. J. Poynton and Dr. A. Paine, "The Etiology of Rheumatic Fever," *The Lancet*, 1900, vol. ii., pp. 861 and 932.

Treatment. Absolute rest in bed is necessary. It is a good plan to fold children in blankets. The diet is that for pyrexia (§ 392). As regards drugs, salicylate of soda or salicin, first adopted by Dr. Maclagan, is rightly regarded as a specific. In most cases it brings down pain and fever within a week, but the treatment must be continued for at least another week, else the symptoms will relapse and the pain return. The drug must be given in large doses—20 grains every 2 hours during the first day or two, then every 4 or 5 hours till the temperature subsides or physiological symptoms of the drug ensue, viz., headache, deafness and buzzing in the ears, albuminuria, or delirium. If initiated early and before cardiac or other complications have arisen this treatment is certain to relieve (Fig. 124). In a few cases, however, especially those in which the joints appear to be less involved than the heart, and those in which the temperature is very high, salicylates may fail. Then antipyrine or antifebrine are remedies of considerable value; in any case they relieve the pain and may be good for this purpose alone; morphia should be avoided. Alkaline carbonates used to be given in large doses, and by some they are still given in combination with salicylates, but Dr. Alexander Haig[1] states that they hinder the effect of the salicylate. The joints should be swathed in cotton wool. An alkaline lotion containing opium (F. 35) may be used if any local application is necessary. If, in spite of the salicylates, the temperature remains high, quinine (10 grains every 2 hours till symptoms arise) may be given. Drugs failing, and the temperature being over 104·5°, a graduated bath should be immediately given, as the condition of the patient requires prompt and energetic measures. If heart complications arise iodides should be given, and many recommend small blisters (the size of a florin) to be applied over the left upper chest or over the heart itself. During convalescence prophylactic treatment is required, because of the liability to relapses and second attacks. The patient should always wear flannel, avoid exposure, and be careful in his diet (see chronic rheumatism).

III. **Acute Blenorrhagic Arthritis** (acute gonorrhœal rheumatism or arthritis) is an acute arthritis resembling "rheumatic fever," due not to the rheumatic diplococcus but to the absorption of the gonococcus or its toxins from the urethra during the acute stage of gonorrhœa. It is far more frequently met with in the chronic form described in § 440. If the disease arises in the acute stage of gonorrhœa the joint mischief resembles acute rheumatism in all respects excepting—(1) although the mischief spreads from joint to joint those first involved do not get better as the others become involved (no metastasis); (2) the temperature has more of an intermittent character than ordinary acute rheumatism; (3) it does not yield to salicylates but runs a prolonged course of many weeks or months; and (4) there is less tendency to heart complications. The joints rarely suppurate, but the disease is most intractable and may lead to extensive adhesions and distortions of the various articulations. It may arise in association with any blenorrhagia, and according to Dr. G. F. Still is by no means unknown in children.

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[1] Haig, "Uric Acid," 2nd Edn., pp. 37—41.

IV. **Pyæmia** has already been described in § 384. In some cases of acute general pyogenic infection the joints are not at all involved (septicæmia), but in others of a pyæmic type there is a marked tendency to a suppurative inflammation in and around the joints. It is differentiated from other joint lesions by:—(1) the swelling does not shift its position, as rheumatism does; (2) the joint may be red and show evidences of suppuration, though this fact may not be revealed until after death; (3) the constitutional symptoms are very characteristic, especially the wide and irregular range of temperature and the rigors and sweatings; and (4) some cause may be revealed in the shape of an internal or external pyogenic focus (p. 646).

V. Other **acute specific diseases** may — though less frequently than the foregoing — lead to inflammation of joints. The joint mischief can be identified only by the presence or history of the disease which it complicates. In *adults* pneumonia and enteric fever may be complicated or followed by septic affection of the joints, often with a fatal issue. Other acute specific fevers may be more rarely so complicated. In dengue joint swelling is often part of the disease. Cerebro-spinal meningitis is almost always accompanied by synovitis. In *children* scarlatinal or diphtheritic synovitis are very common, especially when there have been severe faucial symptoms. The lesion may be suppurative.

Measles, enteric, and influenza are rarer causes in children. Synovitis sometimes follows the administration of antitoxins.

VI. The three remaining generalised disorders associated with joint trouble are notable in that they are all blood disorders accompanied by some morbid condition of the skin, viz., Purpura Rheumatica, Scurvy, and Hæmophilia.

§ 436. **Purpura Rheumatica** (syn. Peliosis Rheumatica, Schönlein's Disease; Henoch's Purpura is probably a variety of the same). In this disease a synovitis resembling rheumatism is associated with a purpuric or erythematous eruption.

Symptoms. (1) Many joints are affected with considerable pain and swelling; (2) the temperature varies between 100° and 103°, the pyrexia usually preceding the arthritis by a day or two, and being accompanied by more or less sore throat; (3) the eruption, which usually starts upon the legs near the joints, is of a purpuric, urticarial or erythematous character. It is attended by a good deal of anæmia. The patient usually recovers in the course of a few weeks, but the disease is apt to recur. The throat symptoms may lead to sloughing of the uvula. Males are more affected than females, and at an age between 20 and 30. The blood change which underlies this condition is unknown; it is probably of rheumatic origin.

In regard to *treatment*, not much is known as to the appropriate remedies, but it would be worth while trying large doses of calcium chloride. Anti-rheumatic treatment is the line usually adopted.

Henoch's Purpura resembles purpura rheumatica very closely. It is chiefly met with in children, is characterised by recurrent attacks of slight pain and swelling of the joints, cutaneous lesions (for the most part erythematous), gastro-intestinal crises, and hæmorrhages from the mucous membranes.

In **scurvy** (§ 411) non-suppurative joint swellings occur; generally not more than one or two joints are affected. The disease is recognised by the spongy bleeding gums, anæmia, and other symptoms of scurvy (*q.v.*).

In **hæmophilia** (§ 412) the larger joints are usually affected. The joint lesion is probably always due to the extravasation of blood or blood serum into the joint cavities, and usually supervenes suddenly on a slight blow or exposure to chill. The joint trouble not infrequently recurs and may ultimately lead to ankylosis. It is diagnosed mainly by the history of hæmorrhages in the patient. The condition is met with for the first time most often between the ages of 7 and 14.

VII. **Acute Traumatic Synovitis** is recognised by the history of an injury, though one must bear in mind (1) that many constitutional processes, especially gout, are lighted up by a very slight injury, and (2) that in childhood, the history of a traumatism may be wanting.

VIII. **Extension** from epiphysitis or osteomyelitis (§ 445) or other bone disease in childhood—set up very likely by injury—may produce acute inflammation in a joint, and the serious nature of the condition may be overlooked unless the correct meaning of the pyrexia and constitutional disturbance is appreciated.

b. *Chronic Joint Diseases.*

Joint disorders which may be chronic *ab initio* come clinically under nine headings.

I. Chronic gout.
II. Chronic rheumatism.
III. Rheumatoid arthritis.
IV. Chronic blenorrhagic arthritis.
V. Other chronic septic disorders.
VI. Tuberculous joint disease.
VII. Syphilitic arthritis.
VIII. Hysterical joint affection, which is often in reality a muscular stiffening and immobility.
IX. Neuropathic arthritis (Tabes, Syringomyelia and Raynaud's disease).

Clinically many of these joint diseases resemble each other very closely both in their physical signs and their history, and many cases are met with which it is almost impossible to place definitely under one or other disease. Moreover, in their pathology we find the same resemblance, for with the possible exception of hysterical and neuropathic arthritis they are all due to some kind of blood change of microbic or metabolic origin.

§ 437. I. **Chronic Gout** usually supervenes upon a succession of acute attacks (§ 434); occasionally it is chronic or sub-acute from the beginning. The joint is stiff and painful on movement, is very tender, sometimes red, and sometimes masses of urate of soda (chalk stones) can be seen through the skin. The patient, who is usually a male over middle age, suffers also from gouty dyspepsia, irritability of temper, and frequent sub-acute exacerbations of joint trouble. The urine may contain a little albumin from time to time. The arteries are generally thick, and there is a marked tendency to high arterial tension.

The *diagnosis* between chronic rheumatism and chronic gout is by no means easy. In an infirmary, where a large number of both diseases in the chronic form are always to be seen, it is usually impossible to classify more than one-third of them. In general terms, chronic gout attacks the smaller joints, the patient is of a plethoric type, and there are concurrent symptoms such as tophi in the ears, interstitial nephritis, or the history of typical paroxysms, which give us some indication of gout. The serum test of acute gout is not of great assistance in the chronic disease (see also table, § 438).

The *prognosis* of chronic gout is more serious than that of chronic rheumatism, though in both the same crippling of the joints occurs. Interstitial nephritis (granular kidney) is almost sure to supervene sooner or later, and the prognosis mainly depends on three factors:—(i.) the condition of the kidneys; (ii.) the degree of arterial tension; and (iii.) the condition of the heart, especially of the heart wall. The complications in addition to those mentioned under acute gout are:—(1) bronchitis, (2) iritis and sclerotitis; and (3) deposits of urates not only in the conjunctiva but in any other tissue of the body. Urethritis may occur in males; stone is not uncommon, and sometimes glycosuria is seen. The patient may eventually die with uræmia, pericarditis, pleurisy, peritonitis, meningitis, or apoplexy. The *treatment* is described under acute gout.

§ 438. II. **Chronic Rheumatism** is an extremely common affection of the joints. The disease may follow one or more acute attacks, or as is more usual come on insidiously as a chronic affection from the beginning. There are some grounds for believing that chronic rheumatism is pathologically distinct from acute rheumatism. The capsule, ligaments and tendon sheaths are thickened. The joint is stiff and creaks with adhesions, is generally more or less swollen and sometimes tender. Sometimes many joints are affected symmetrically; sometimes only one is affected. Ultimately the joint may be considerably distorted, but not disorganised, there being a tendency towards adhesion and fibrous thickenings. When the hands are affected they are in time permanently deformed. The general health, in many cases, is not disturbed, and there is no tendency to either heart or renal affections; though there is a certain amount of anaemia and a variable degree of pain. The disease is never fatal.

The *diagnosis* from rheumatoid arthritis and chronic gout is sometimes very difficult, and may be impossible until the later stages are reached, when rheumatoid arthritis has "lipping" or out growths of the bone (see table below). Chronic rheumatism is sometimes known as "poor man's gout" because of its tendency to occur in the poor and destitute and in those whose occupation exposes them to cold and damp.

Apart from arthritic heredity the *etiology* is obscure. The patient is generally past middle life, excepting where chronic rheumatism has followed acute attacks, when the patient may be younger.

TABLE OF DIAGNOSIS.

Chronic Rheumatism	*Rheumatoid Arthritis*	*Chronic Gout.*
Either sex: middle life or over.	Chiefly female sex: any age, but often under 35.	Male sex only: over 40.
Poor and debilitated.	Poor and debilitated.	Rich and plethoric.
Insidious onset, unless following rheumatic fever.	Insidious onset, but little pain. Skin cold, blue and clammy.	History of sudden onset and acute attacks with severe pain. Skin over joints red, swollen, and œdematous.
Generally poly-articular. Temporo-maxillary not affected.	Symmetrical affection of the smaller joints usual. May sometimes be mon-articular. Temporo-maxillary joint often affected.	Only one joint affected at first.
Thickening of tendons and ligaments — no bone changes.	Ineradicable hypertrophy and "lipping" of bone ends. Heberden's nodes near finger joints.	Deposits of urate of soda round the joints.

Treatment. Drugs are not nearly so potent in chronic as in acute rheumatism; among those which may be found useful are potassium iodide, guaiacum, quinine, alkalies, sarsaparilla; and in cases with much weakness and anæmia, cod-liver oil is excellent. Local treatment is often more useful, *e.g.*, iodine, blistering, turpentine or other liniments, actual cautery, massage, Scott's dressing, or the application of oleate of mercury. The diet is of considerable importance (§ 214). The patient should avoid sugar and alcohol of all kinds, excepting in very debilitated states, when a small quantity of spirits may be taken. What is known as the Salisbury treatment is certainly very efficacious in some cases (p. 376).[1] One of the most satisfactory methods of treating chronic rheumatism and many other joint affections is the application of hot air. This may be given in the form of Turkish baths, or in the form of superheated air as given in the Tallerman method,[2] or by radiant heat. On the same principle passive hyperæmia of the joint, induced by bandaging firmly below and above, has given good results. Baths of many different kinds have been used, chiefly sulphur and alkaline baths—see F. 1, 3, and 6. Change of climate does much for those who can afford it. Wintering abroad to avoid cold and damp is undoubtedly most beneficial, especially in a climate like Egypt. Climatic treatment may be combined with baths, as at Aix-les-Bains, Baden-Baden, Buxton, Bath, Carlsbad, Contrexeville, Harrogate, Mont-Dore, Strathpeffer, etc.

§ **439.** III. **Rheumatoid Arthritis** (Arthritis Deformans) consists of a degeneration and destruction of the joint cartilages, attended by bony outgrowths, leading to great deformity.

Symptoms. Four varieties of the disease are recognised:—(1) general progressive; (2) Heberden's nodes; (3) mon-articular; (4) arthritis deformans of children.

(1) *The general progressive* variety occurs in *acute* and *chronic* forms. (a) The acute form is met with chiefly in young women, and has slight pyrexia. Many joints, especially the finger joints, are affected; they are pale, swollen, tender, the fingers having a

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[1] A very satisfactory case of this treatment is published in *The Lancet*, 1893, vol. ii., p. 133.

[2] This is applied in the following way:—the limb is placed in a specially-made copper chamber made to fit the limb, and the temperature in the interior is gradually increased up to 250° or 300° F. Each application lasts about 20 minutes. The anodyne effect is said to be remarkable, and permanent benefit is reported in some cases.

characteristic spindle-shaped appearance. The swelling does not shift from joint to joint as in acute rheumatism. (*b*) The chronic progressive variety is the most common form. The onset is insidious, and is accompanied by anæmia and debility, and often menorrhagia and leucorrhœa. The earliest joint signs are pain and creaking on movement, and swelling, rarely redness: periods of slight improvement alternate with periods of pain and swelling. Ultimately the shape of the affected joint is altered by the formation of osteophytes, thickening of the ligaments and atrophy of the muscles. Great deformity, locking of the joints and contractures ensue, and the fingers are deflected to the ulnar side (Fig. 125).

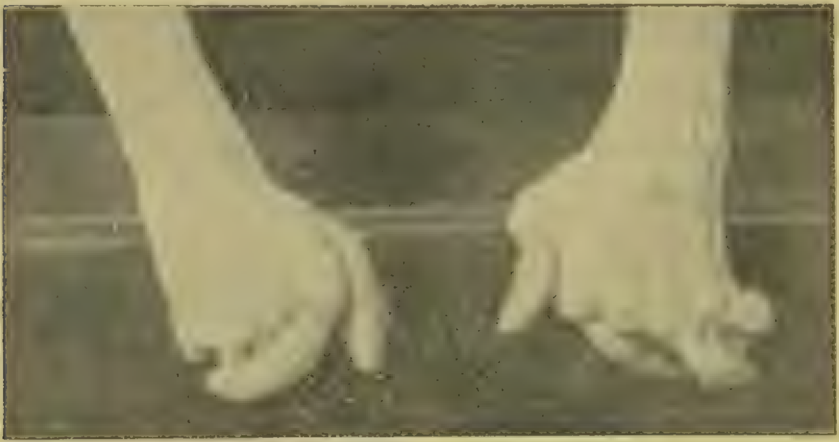

Fig. 125.—Rheumatoid Arthritis in a man about 38 years of age, showing ulnar deflection of fingers.

(2) In "Heberden's nodes" the finger joints show hard knobs at each side due to lipping or bony outgrowths.

(3) The monarticular form occurs in old people, and especially affects the knee, hip, shoulder or spine. In the vertebral form there is progressive ankylosis of the spine.

(4) A form occurring in children before the second dentition has been described, associated with enlargement of the glands and spleen, slight pyrexia and anæmia.

The *diagnosis* from chronic rheumatism is very difficult, and until the bony outgrowths can be felt, almost impossible: the chief points are given in the table in § 138. Chronic rheumatism is said not to attack the temporo-maxillary joints. If this joint is affected the disease is probably rheumatoid arthritis or blenorrhagic arthritis.

Prognosis. The disease is very chronic and incurable, but presents no danger to life. In the chronic progressive form the joints of the hands are usually first affected; and the disease may then remain quiescent; in other cases all the joints of the body may be attacked and the patient may suffer extreme agony. Pigmentation, numbness, glossy skin and other trophic changes may occur.

Etiology. Rheumatoid arthritis is apt particularly to affect the female sex, and to start between the ages of 25 and 40. The mon-articular form, however, usually affects elderly men. Mental worry or grief and other diseases of neurotrophic origin have been said by some to predispose. The author believes it to be a chronic toxæmia, possibly of intestinal origin.

The *treatment* is much the same as that for chronic rheumatism, but massage seems to do harm, excepting when applied to the atrophied muscles. Another point in which this affection differs markedly from chronic rheumatism is in the dietetic treatment. Instead of being light, it should be liberal and generous. Drugs are not of much use; salicylates occasionally relieve the attacks of pain. Cod-liver oil, malt, iron and arsenic are in my experience the best remedies. Liniments, especially of chloroform and belladonna, and electrical baths have proved useful in some cases. I have known of three very chronic cases which improved remarkably under the combined current with three weeks' rigid "Salisbury" diet, followed by a very generous diet. High frequency currents do good in some cases.

§ 440. IV. **Blenorrhagic Arthritis** (syn. gonorrhœal rheumatism, urethral arthritis) is a synovitis associated with a gonorrhœal or other urethral discharge, resembling chronic rheumatism in some respects, chronic pyæmia in others. An acute form has been referred to on p. 794, but the disease is nearly always chronic. In this, the chronic and commoner form, the joint affection comes on insidiously during a gleet (often about the fourth or fifth week) or any kind of blenorrhagia. According to surgeons who see many of these cases, it is particularly apt to supervene on those gonorrhœas in which the prostatic portion of the urethra is affected, and the extreme vascularity of that part would lend probability to this fact. When the joint becomes involved the gleet sometimes disappears, and this may give rise to an error in diagnosis. In the chronic variety there is only slight elevation of temperature of an intermittent or septic kind; but there is always more or less disturbance of the general health; and this may be very great, the patient becoming anaemic and emaciated. Some say that the affection is usually mon-articular, the knee being its favourite seat; but in all the cases I have seen many of the joints have become progressively involved. It never shifts its position, but is progressive. It is apt, moreover, to attack many of the joints exempt from other diseases, such as the sacro-iliac, sterno-clavicular, and temporo-maxillary. The affected joint becomes swollen, stiff and tender, and gradually becomes permanently damaged, resulting perhaps in ankylosis or dislocation. The fibrous tissues also are often affected, especially the plantar fascia; pain in this position or the tendo achillis is an important diagnostic feature of the disease.

For the *diagnosis* one has to rely mainly on the history of blenorrhagia, the inveterate character, the tendency to ankylosis, and the fact that it is wholly unrelieved by salicylates.

The *prognosis* as regards life is favourable, but not as regards recovery. The heart is seldom affected, but there may be pleurisy or iritis. It is more hopeful in younger people and in attacks of recent date. The joint mischief may go on for months or years. The probability of cure depends a great deal on the curability of the urethritis.

As regards *etiology*, both men and women may be affected. The nature of this malady has never been actually determined, but there is every reason to believe that it is of an infective nature, due to the absorption of some toxin from the urethra. It has been definitely shown that the gonococcus may be present in the joint, with or without strepto- or staphylococci. Special exposure to chill during a gonorrhœa will sometimes determine the disease.

Treatment. The first indication is to cure the urethritis. This is sometimes extremely difficult, especially when, as generally happens, the gleet takes on a fresh development after its temporary disappearance. The gum resins and ordinary injections are useless in most cases. Urotropin, helmitol and other urinary antiseptics may be tried. For the joint mischief one of the most useful applications is Scott's dressing with ung. hyd. or oleate of mercury; and the treatment suggested for chronic rheumatism may be tried. Mr. Christopher Heath[1] has treated cases with belladonna and glycerine applications, and 5 grains of sulphate of quinine every 6 hours internally; and he states that the patients were usually better in 3 or 4 weeks. Iodide of potassium in large doses is recommended by some. A sea voyage is in my experience one of the most efficacious remedies. Climatic and bath treatment is also advantageous. It is of the greatest importance to improve the patient's general nutrition. The patient should avoid thereafter any possibility of a fresh attack of gonorrhœa and blenorrhagia, as this would most certainly be followed by a recurrence of the joint symptoms. Surgical treatment by irrigation of the joint in severe cases is often efficacious.

V. Other forms of **chronic septic arthritis.** Chronic infective arthritis (chronic pyæmic arthritis) is not a form of chronic joint affection generally recognised by authors, so far as I am aware, unless the chronic form of gonorrhœal rheumatism is so regarded. A case which I had under my care for many months (and one other of a similar nature) is described and illustrated on p. 650, which is best explained on the supposition that the joint mischief was due to the absorption of septic matter from an internal pyogenic focus, namely in the appendix. Cases of arthritis have been recorded[2] which occurred during convalescence from dysentery.

§ 441. **Tuberculosis, Syphilis, Hysteria,** and other **nervous disorders** also affect the joints.

VI. **Tuberculous Joint Disease.** Tuberculosis affects chiefly the synovial membrane, but it may commence in the articular ends of the bones. This is *par excellence* the mon-articular joint disease of children.

Symptoms. The onset is extremely insidious, though not infrequently the symptoms date, or are supposed to date, from an injury. The favourite situations are hip and knee-joint, though any joint may be affected. The child may complain of slight pain, which gives rise to limping, for weeks or months before anything is apparent. Generally the disease is in the knee, but sometimes it is in the hip, although the pain may still be referred to the knee, the nerve supply of which is a branch of the obturator nerve. By and by the affected joint swells, it is pale and has a pulpy or doughy feel beneath the finger, and fluctuation may be felt. If untreated, the case goes on to abscess formation. The constitutional symptoms consist of an intermitting pyrexia and general debility which are present even from the very beginning.

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[1] *The Lancet*, Nov. 25, 1899, p. 1467.

[2] Dr. Paul Remlinger, *Revue de Médecine*, Sept. 1898, p. 685.

The *causes* are the same as those mentioned under phthisis. The symptoms may date from or be first noted after an injury. The disease nearly always attacks children, though a more destructive form of tuberculous joint mischief does occur in advanced life. The disease may last for many years, and the prospect of recovery depends very much upon the stage at which it first comes under treatment. If this is neglected extensive destruction of the joint may occur, and very frequently tuberculous mischief is found in other organs. The *treatment* is mainly surgical, but a good deal can be done in the early stages by rest and constitutional treatment.

VII. **Syphilitic Joint Disease.** Syphilis has of late years been recognised as a cause of chronic joint disease. The differential features of this form of arthritis are as follows. (1) One or several joints may be affected. The synovial membrane may be attacked, leading to a doughy swelling, or the ligaments or cartilage. There is no "lipping" of the bone ends. (2) The joint manifests no signs of acute inflammation; occasionally some effusion. (3) The pain is characteristic in being very moderate during the day, but subject to nocturnal exacerbations. (4) Other evidences of syphilis are generally present. (5) All but one of the cases I have seen have been in children. (6) The condition is very chronic and is only partially amenable to iodides.

A PSEUDO-PARALYSIS of SYPHILITIC ORIGIN occurs in children, due to the separation of the cartilage from the diaphysis, which is apt to be mistaken either for joint disease or for infantile paralysis. The affected part is however acutely tender.

VIII. **Hysterical Joint Disease** usually affects the hip or the knee, and it often dates from some trifling injury. The joint is fixed, tender (often more tender to light touches than to deep pressure) and sometimes swollen; and the local temperature of the joint may also be raised. Sometimes there are no physical signs referable to the joint at all. The loss of function may be entirely due to muscular rigidity, and in the case of the hip-joint the condition may very precisely resemble (mimic, as Sir Jas. Paget[1] says) tuberculous disease of this joint. The *diagnosis*, which is often extremely difficult, rests mainly on (1) the absence of serious mischief in the affected joint when examined under chloroform, (2) the disproportionate loss of function, (3) the patient being a female and the subject of other manifestations of the hysterical diathesis.

The *treatment* should be mainly directed to the hysterical diathesis (*q.v.*). The joint mischief may sometimes be cured by the anæsthesia of chloroform; on coming round the patient finds that she can use the joint, and continues to do so. In the author's view[2] these cases are due to a vascular change in the synovial membrane probably of vaso-motor or toxic origin, albeit slight and perhaps temporary and evanescent. Many cases of undoubted hysterical joint disease are amenable to salicylates, or alkaline carbonates on the one hand, or to bromides or vaso-motor remedies on the other.

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[1] "Lectures and Essays," edited by Mr. Howard Marsh, London, 1879.

[2] "Lectures on the Pathology of Hysteria," *The Lancet*, Jan. 20, 1904, and *Clinical Journal*, May and June, 1904.

IX. **Neuro-trophic Arthritis** (neuro-arthropathy, Tabetic Arthropathy, Arthritis in connection with spinal lesions). Two diseases of the spinal cord are sometimes, though comparatively rarely, attended with chronic mischief in the joints, viz., *tabes dorsalis*, and *syringomyelia*. In both it may occur in an early stage of the disease, when nervous symptoms are few or absent; and in both extensive disintegration of the joint may take place, without pain, heat, or redness, and without giving rise to much inconvenience. In *tabes dorsalis* the associated joint lesion is known as tabetic arthropathy or **Charcot's joint disease**, because it was he who first identified the connection. This lesion may occur without the patient suffering any pain, and but little inconvenience, although the bone ends may be enlarged, and it may go on to extensive disorganisation with increased mobility and new bony formations before the patient seeks a doctor's advice. A case of *tabes dorsalis* is narrated by Prof. J. M. Charcot[1] of a soldier, in whom actual dislocation of both hips was found to have occurred without the patient being aware of any mischief in the joints. Indeed, it had happened while he was on the march. In all such cases the pupils and knee jerks should be examined. The knee is the favourite situation, and therefore it is sometimes difficult to test the tendon reflexes.

Syringomyelia is characterised by muscular atrophy and anæsthesia at the ends of the extremities. Any joint may be involved; in two cases I have seen very extensive mischief affected the joints of the upper extremities.

In *Raynaud's disease* a sub-acute or chronic synovitis sometimes occurs which is possibly of vaso-motor origin.

GROUP III. MUSCULAR DISEASES.

We are here concerned with lesions situated in the muscular substance as evidenced by pain localised in the muscle (myalgia) and tenderness, accompanied perhaps by some swelling. The causes of pain in the limbs were discussed in § 423. The causes of muscular weakness will be dealt with in the chapter on nervous diseases.

I. Muscular rheumatism or gout.
II. Neoplasms.
III. Trichinosis.
IV. Idiopathic myositis.

§ 442. I. **Muscular Rheumatism** is certainly the most frequent cause of muscular pain and tenderness in this country. It is difficult, if not impossible, to separate gouty from rheumatic muscular inflammation. *Symptoms.* (1) The pain usually comes on quite suddenly; so suddenly indeed, in the case of lumbago, that it is often mistaken for a sprain or rupture of the muscular fibre. It is greatly aggravated by movement and relieved by rest. In the more acute cases it is attended by localised tenderness. (2) Little or no swelling can be detected in the affected muscles, a point of distinction from trichinosis and new growths. (3) It is usually accompanied by a furred tongue and disordered digestion, with constipation and a copious deposit of lithates in the urine. There may be slight pyrexia.

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[1] "Nouvelle Iconographie de la Salpêtrière," about 1887.

The commonest *variety* of muscular rheumatism is **lumbago**, where the pain is situated in the muscles and fascia of the small of the back. It is usually of very sudden onset, often when in the act of stooping. Rheumatic torticollis is a rheumatic affection of the sterno-mastoid, and is met with chiefly in children. Intercostal rheumatism is a similar affection of the intercostal muscles. Lumbago has to be *diagnosed* from other causes of lumbar pain (p. 473). In aneurysm of the dorsal aorta the pain is more continuous, not so easily relieved by muscular rest. In myelitis and meningitis there are other symptoms referable to the nerve trunks, sensory or motor. Muscular rheumatism though not lethal is very painful and incapacitating, and is very prone to recur.

Etiology. Muscular rheumatism mostly arises in gouty and rheumatic subjects who present other evidences of lithæmia (§ 251). It is usually determined either (i.) by a chill, especially after prolonged exertion accompanied by profuse perspiration, or (ii.) a muscular strain. It comes on especially in cold and damp weather. Cold or damp alone do not seem able to produce it; it is when the two occur together, and especially when combined with errors of diet, that the disease is chiefly produced. Sugar, rich foods, and sweet heavy wines are most potent for evil. Prof. Ralph Stockman[1] has found fibrous nodules in the muscles, tendons, fascia, and nerves in chronic rheumatism, which swell and become more painful in cold damp weather.

Treatment. The treatment must be directed on the lines laid down for gout and rheumatism in other parts, and consists mainly of free purgation with calomel and salines, of alkalies, sodium salicylate, or guaiacum (F. 96); quinine and iodide of potassium are useful in protracted cases. Rest is necessary for the pain; and even morphia may be required. Locally, counter-irritants are best. Lint soaked in a mixture of equal parts of liniment of belladonna and chloroform should be kept over the muscles (or in hospitals, liniment of turpentine does equally well and is very much cheaper); it should not be covered up with oil silk or it will blister. Dry heat is very efficacious. The galvanic current allays the pain and promotes recovery in some cases. Warm underclothing and a flannel belt may prevent recurrence. The diet must be very simple (milk chiefly), and sugar and alcohol avoided (see also § 438). Sometimes an attack may be aborted by massage and a Turkish bath. Dr. Stockman (*loc. cit.*) advises that the nodules should be massaged perseveringly, in spite of the pain caused, for 6 or 8 weeks; and that this should be followed by exercises to stretch the muscles and aponeuroses.

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[1] *Brit. Med. Journ.*, February, 1904, p. 477.

II. **New Growths** in the substance of the muscles may give rise to pain and tenderness, usually associated with swelling. The pain and tenderness are in this case strictly localised, at any rate at first, to the seat of the disease: and there is a thickening or tumour discoverable on careful palpation. In some cases, *e.g.*, syphilitic and malignant growths, the lymphatic glands in the neighbourhood are enlarged. The chief neoplasms affecting muscles are:—(a) Innocent—syphilitic gumma; abscess, which may arise from a gumma or be of inflammatory origin; fibroma; lipoma; angioma; hydatid or cysticercus. (b) Malignant—sarcoma; and carcinoma (by extension). First determine whether the swelling is inflammatory or non-inflammatory, malignant (and rapidly growing) or non-malignant, by an investigation of the swelling, the glands, the history, and the concurrent symptoms. The diagnosis and treatment is mainly surgical.

§ 443. III. **Trichinosis** is a disease due to the presence of a nematode worm (the trichina spiralis), in the intestinal canal, and the dissemination of the embryos in the blood and the muscular system, consequent on the ingestion of "measly" meat (usually pork) insufficiently cooked. It is rarely met with in this country.

The female *adult* or intestinal worm measures about $\frac{1}{8}$ inch, the male slightly less. In fæcal examinations for the parasite it should be remembered that the characteristic feature is the "cell body" at the anterior part of the intestine of the parasite. The *larvæ* (Fig. 126) or muscle trichinæ are found in infected muscle, where they are visible by the aid of a $\frac{1}{2}$ or 1 inch lens. Each consists of an ovoid capsule (translucent, or infiltrated with lime salts, according to the length of time it has existed) containing two or more embryos coiled up within it. The *embryos* are 0·6 to 1 mm. long, with pointed head and rounded tail. It is the presence of these larvæ which give to pork or other infected meat a characteristic "measly" appearance which is visible to the naked eye. Trichina has been found in the flesh of 26 different species of animals, but it is chiefly conveyed to man by "measly" pork, insufficiently cooked; the capsules are then digested, and the embryo set free in the intestinal canal of the patient. During the ensuing week the embryos grow and attain sexual maturity; each female being capable of producing several hundred embryos. After fecundation the female worm penetrates the walls of the intestinal canal, and hundreds of embryos are discharged into the lymph spaces, or directly into the blood stream, and are carried chiefly to the muscles, where after 2 or 3 weeks they become encysted and assume the larval form. In the course of months or years the larvæ become calcified, but they have been found to be alive and capable of developing 10 or more years after their entrance.

Symptoms. The disease runs a course of several weeks, and consists of three well-marked stages. The *first* stage, before the larvæ begin to migrate, lasts usually about a week or 10 days, during which the symptoms are those of gastro-intestinal disturbance attended by abdominal pain, with more or less diarrhœa and vomiting. It is important to discover the disease in this stage as treatment is so much more hopeful, and the adult worm should be sought for in the stools. The *second* stage, which lasts from 2 to 3 weeks, is coincident with the active migration of the embryos. This gives rise to acute universal muscular pain, tenderness, and sometimes swelling. The wandering of the embryos in the muscles produces a shortening and rigidity; the biceps seem specially apt to be affected, resulting in a typical flexion. In severe cases, movement of the affected muscles, *e.g.*, turning the eyeball, moving the limbs, chewing, swallowing, etc., aggravates the pain. Pyrexia of a remitting or intermitting type, is present, with profuse perspiration, inability to sleep, and possibly delirium. In some cases there is extreme dyspnœa from impairment of the diaphragm. There may be general œdema, starting in the face, and later on, emaciation. In slight cases the muscular and other symptoms may be so insignificant as to be altogether overlooked. In the *third* stage the acute symptoms gradually subside, great muscular weakness ensues, and recovery is slow. This stage is apt to be interrupted by various complications, especially pneumonia, pleurisy, or persistent and intractable diarrhœa.

In slighter cases the *diagnosis* from muscular rheumatism or other diseases in this group may present difficulty, though the widespread muscular tenderness of trichinosis, the history of gastro-intestinal symptoms, and the epidemic occurrence in a whole family should aid us. The stools after a large dose of calomel may be searched for the adult worm; and it has been suggested to remove a small portion of muscle by means of a harpoon for microscopic examination. Some cases are mistaken for enteric fever and *vice versâ*. In trichinosis, however, Dr. T. R. Brown has shown that there are characteristic and distinctive changes in the blood, in the shape of a very marked leucocytosis—reaching 30,000 per cmm. or more—due mainly to an enormous increase in the eosinophil cells, which may amount to 50 per cent. of all the leucocytes as compared with the normal 2 to 5 per cent.

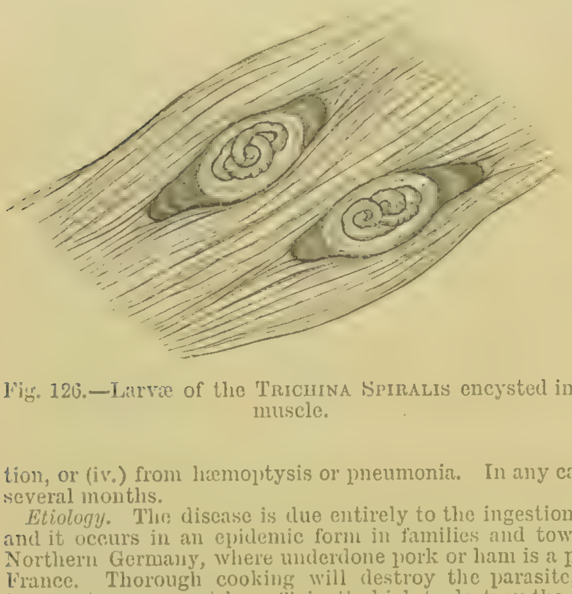

Fig. 126.—Larvæ of the TRICHINA SPIRALIS encysted in muscle.

Prognosis. There is every grade of severity from slight muscular pains to profound muscular and constitutional disturbance, depending on the degree of infection, *i.e.*, the amount of meat eaten and the amount and age of its contained larvæ. The disease not infrequently ends fatally between the 3rd and 6th week; the mortality varying from 2 per cent. to 30 per cent. The intensity and duration of the symptoms is a fair measure of the prospect of recovery. Death may occur from (i.) diarrhœa, (ii.) asphyxia (from involvement of the respiratory muscles); (iii.) from exhaustion, or (iv.) from hæmoptysis or pneumonia. In any case health may not be restored for several months.

Etiology. The disease is due entirely to the ingestion of "measly pork" or other meat; and it occurs in an epidemic form in families and towns. It is much more frequent in Northern Germany, where underdone pork or ham is a popular food, than in England and France. Thorough cooking will destroy the parasite, but in large joints the cooking temperature may not be sufficiently high to destroy the parasite in the interior. All meat, particularly sausages and pork, should be thoroughly well cooked. It is said that 170° F. will destroy the larvæ, but that 107° F. will do if maintained long enough. This temperature must permeate to the interior of the meat.

Treatment. If the patient is seen within 2 or 3 hours after the ingestion of infected meat an emetic should be given. If the disease is discovered within 24 or 30 hours, the gastro-intestinal tract must be thoroughly cleared out. Glycerine in large doses has been recommended in the first stage for its hygroscopic properties to destroy the nematode. Filix mas, kamala, santonin, thymol and turpentine are also recommended. If, however, the second stage is reached and the embryos are migrating, the treatment must be symptomatic, because nothing will destroy them. For the pain and tenderness, opium and other anodynes may be required.

§ 444. IV. **Myositis** or inflammation of the voluntary muscles is an extremely rare condition, and only a few cases have been placed on record. Three forms are recognised by authors:—(a) a *localised* form, in which pain, tenderness, swelling and impaired movement are localised to one muscle or a group of muscles; (b) an *acute generalised* form in which these symptoms, accompanied perhaps by œdema and redness of the skin, are more widespread; (c) a *progressive generalised* form of myositis in which the disease runs a prolonged course spreading from muscle to muscle. An example of this variety is reported by Dr. J. K. Fowler.[1] The disorder began in the left thigh, and in about two years became general. It was characterised by painful knotty swellings and followed by general wasting of the muscles. In *myositis ossificans* the process goes on to the formation of bone.

The *diagnosis* of myositis appears to offer some difficulty, especially from trichinosis. A microscopic examination by means of a harpoon would afford aid in the acuter cases; and the prolonged progressive course of myositis aids the diagnosis of chronic cases.

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[1] Clin. Soc. Trans., April 23, 1897.

The *causes* of idiopathic myositis are very obscure. It seems possible that syphilis may have been in operation in some cases. The acute localised form is generally the result of injury, or spread from surrounding structures. Some think that the generalised varieties are essentially of nervous origin. Dr. F. Parkes Weber, who has kindly supplied me with several references,[1] regards such cases as neuro-dermatomyositis.

In regard to *treatment*, iodides may be tried. In Dr. Fowler's case the only treatment which proved of service consisted of warm baths, which relieved the pain and stiffness.

GROUP IV. BONE DISEASES.

It would be out of place to deal at any length with diseases of the bones, which belong in a special manner to the surgeon. Nevertheless, these diseases frequently come under the notice of the physician, especially in their early stages. Pain and deep-seated tenderness are often their chief and sometimes their only symptom. Pyrexia and constitutional derangement may be present. Deep-seated swelling and deformity may appear later, and if the bone is superficial, œdema and redness of the skin. The majority of bone diseases are chronic (p. 810).

Acute Bone Diseases.

I. Acute osteomyelitis.
    Acute periostitis.
    Acute epiphysitis.

Chronic Bone Diseases and Deformities.

I. Rickets.
II. Chronic periostitis, osteitis, caries, and necrosis.
III. Tumours of bone.
IV. Acromegaly.
V. Achondroplasia.
VI. Pulmonary osteo-arthropathy.
VII. Osteitis deformans.
VIII. Mollities ossium.
IX. Leontiasis ossea.

§ 445. Acute Infective Osteomyelitis (syn. acute necrosis; acute periostitis). This disease, which used to be known as acute necrosis, and later as acute periostitis, is an acute inflammation affecting one of the bones, starting in the medulla or periosteum, rapidly involving the whole bone, accompanied by severe constitutional disturbance—on which account the case comes under the notice of a physician. Osteomyelitis is the only really acute bone disease, though acute symptoms very closely resembling those of osteomyelitis may arise in association with a LOCALISED PERIOSTITIS such as results (especially in children) from an injury.

The *symptoms* of acute osteomyelitis are—(1) pain of a very severe character coming on suddenly, and attended by extreme tenderness, starting usually at the articular end of the bone—very often the tibia—attended in the course of a day or two by swelling of the limb, at first pale, and afterwards red, as the inflammation makes its way towards the surface. (2) The constitutional symptoms come on suddenly and are very marked. The temperature is high, and there are rigors and great prostration. The *diagnosis* from acute rheumatism, which it may at first resemble, because of the pain starting near a joint, is made by the fact that in rheumatism the pain and swelling are confined to the joint, by the early involvement of other joints, and by the profuse perspiration. Acute epiphysitis is mentioned below. The *prognosis* is always very grave. If the patient survive the initial constitutional disturbance the malady leads to necrosis and tardy convalescence. The most frequent complications are septicæmia and the extension of the inflammation to a joint. *Etiology.* Acute osteomyelitis is more frequent in children under the age of puberty. It sometimes dates from an injury, though it is a microbic infection. It may occur as a sequel to the continued fevers. *Treatment.* Rest and antipyretics with quinine are indicated, but a surgeon should at once be called in to consider the advisability of free incision, drainage, and other surgical measures.

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[1] (1) Max Levy-Dorn, "Polymyositis and Neuritis," *Berl. Klin. Woch.*, Sept. 2, 1895; (2) E. Wagener, "Ein Foll von Acuta Polymyositis," *Deut. Arch. f. Klin. Med.*, 1887, p. 241; (3) Unverricht, "Polymositis Acuta Progressiva," *Zeit. f. Klin. Med.*, vol. xii., p. 533; (4) Unverricht, "Dermatomyositis," *Deut. Med. Woch.*, 1891, p. 41; (5) Hepp, "Ueber Pseudo-trichinose," *Berl. Klin. Woch.*, 1887, p. 297, etc.; (6) Senator, in *Deut. Med. Woch.*, 1893, p. 933; (7) Senator, in *Zeit. f. Klin. Med.*, 1889, xv., p. 61.

Acute Localised Periostitis may arise from traumatism and if not infected it soon subsides. If infected either from without (a wound), or from within (the blood), suppuration and necrosis take place and the condition becomes chronic (§ 447), which is more common than acute periostitis.

Epiphysitis is inflammation beginning in the cartilaginous layer which in early life separates the epiphysis from the shaft of the long bone. The *acute* form is met with in very early infancy. Suppuration soon sets in and spreads to the joint, forming abscesses. It may resemble acute osteomyelitis, but the profound constitutional disturbance is lacking. It is distinguished from acute rheumatism by the age of the patient and by the development of abscess. In the *chronic* form the process is much slower and is of interest chiefly in relation to the diagnosis of rickets, from which it differs in being localised to one joint. As regards causation the acute form is generally referable to an injury and sepsis; the chronic form is generally associated with syphilis or tubercle.

Chronic Bone Diseases and Deformities.

	Occurrence.	Age period most affected.	Number of bones affected.
Rickets.	Very common.	Infancy (6 to 12 months).	Many bones symmetrically.
Ch. periostitis, osteitis, caries and necrosis.	Common.	Early life.	One bone; may be several in syphilis.
Tumours.	Relatively uncommon.	All ages.	Starts in one bone.
Acromegaly.	Rare.	25 to 40.	Extremities and face.
Achondroplasia.	Very rare.	Congenital.	Whole skeleton.
Pulmonary osteo-arthropathy.	Rare.	After middle age.	Arms chiefly.
Osteitis deformans.	Very rare.	After middle age (men).	Long bones.
Mollities ossium.	Very rare.	25 to 35 (women).	Whole skeleton.
Leontiasis ossea.	Very rare.	After middle age.	Facial and cranial bones.

§ **446.** I. **Rickets** (syn. Rachitis) is a constitutional disorder of childhood attended with epiphyseal enlargements and other deformities of the skeleton. It was described by Glisson in 1675.

The *symptoms* for which we are consulted, coming on between the 6th and 12th month, are—delayed dentition and walking, or the child cannot "sit up"; gastro-intestinal disorders; bronchitis; sweating about the head; or a generalised tenderness and restlessness. In the limbs the disease is typically shown by the *enlarged epiphyses*, affecting most if not all of the long bones. The rib-ends are the first to show the enlargement at their junctions with the costal cartilages and thus produce an appearance of "beading"—the "rickety rosary." The spine has a general backward curvature when the child sits up; scoliosis may ensue later. The head is square shaped, both the frontal and parietal eminences are prominent. The fontanelles may remain open after the 2nd year (normally they should close between 15 months and 2 years of age). There may be craniotabes (thinning of the skull bones) especially of the occipital region. The body may be emaciated or plump and flabby. The chest is "pigeon-breasted" and shows a lateral depression (see Fig. 37, p. 162) and a transverse groove, "Harrison's sulcus." The liver and spleen are both enlarged in advanced cases; the ribs are pushed forward and the belly is prominent.

Diagnosis. The disease may have to be diagnosed from hereditary syphilis in which there may be enlargement of the epiphyses, but this occurs usually only in one bone and is accompanied by other undoubted signs of syphilis. It may also need to be distinguished from the other diseases of this group which affect children. Infantile paralysis soon exhibits muscular wasting. Achondroplasia (§ 448) is a rare condition which has only recently been distinguished from rickets. In infantile scurvy (§ 414) the swellings affect the shaft rather than the epiphysis, and are painful.

The diagnosis of rickets, hereditary syphilis, and hydrocephalus is given in the form of a table on the next page; it is chiefly in regard to the form of the head that the diagnosis between hydrocephalus and rickets presents any difficulty.

Prognosis. The disease when taken in hand before osseous changes are marked is readily amenable to treatment. If untreated

TABLE OF DIFFERENTIAL DIAGNOSIS OF

	Rickets.	*Hereditary Syphilis.*	*Hydrocephalus.*
I. History.	Gastro-intestinal irritation, sweating about head. Improper feeding.	Snuffles and rash. Miscarriages in mother.	Congenital, or may be acquired after some meningeal inflammation, or due to tumour pressing on veins.
II. Age of patient.	Commences in infancy and begins to show itself during the 1st or 2nd year.	Symptoms first appear 3rd week and the 3rd month.	Congenital or acquired.
III. Shape of head.	Often compressed antero-posteriorly. Frontal eminences marked.	Irregular prominence on each frontal and parietal bone. Skull has been called natiform. Depressed bridge of nose.	Bulges in all directions. General tendency to assume a globular form.
IV. Fontanelles.	Close late.	Appear to be depressed in the hollow between the 4 prominences.	Bulging; separation of the bones at the sutures.
V. Other peculiarities.	Epiphyseal enlargements, delayed dentition, etc.	Pegged and notched teeth. Scars about mouth, palate, etc.	Stunted growth, mental deficiency.

it leads to deformity. If death occurs it is due to some complication, notably bronchitis or other respiratory complication, or from gastro-intestinal disorder, laryngismus stridulus or convulsions. Tetany is apt to supervene. Spinal, pelvic and other deformities, or hydrocephalus may result, and the growth is stunted. Genu-valgum (knock-knee), genu-varum (bow-leg), and flat-foot often result.

Etiology. Rickets rarely appears earlier than 6 months or later than the 2nd year, though Sir William Jenner described a late rickets arising up to the 9th or even the 12th year. Dr. Samuel Gee collected 635 cases and found it seldom began later than 12 months, and his latest case was only 20 months old.[1] Both sexes are equally affected. The disease is more frequent in cities, and impure air may play a part in its production, but it may also be found in the country and among the wealthy. Too prolonged lactation, suckling during pregnancy, and too rapid pregnancies in the mother predispose to rickets in the offspring. However, it is now generally admitted that the essential cause of rickets is a defect in the diet. The diets on which rickets is produced all show, according to Dr. Cheadle, a deficiency in two constituents—animal fats and proteids. A deficient assimilation of lime salts also appears to aid. Too early or excessive administration of carbohydrates and particularly the proprietary infant foods rich in starches is in actual practice a frequent cause.

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[1] Quoted by Dr. Hilton Fagge, "Prin. and Pract. of Med."

The *Treatment* must be mainly dietetic (compare children's dietary, § 214a). Carbohydrates must be reduced, and raw meat juice, milk-casein, or other proteid food-stuffs, and good milk and cream added. Cod-liver oil is the best drug to give, either alone or combined with lime preparations and with iron. Phosphorus $\frac{1}{120}$ gr. t. d. in oil is also recommended. One of the best remedies for unhealthy stools is F. 64. The child must not be allowed to walk lest the bones yield and produce permanent curvatures. Fresh air and sunlight are necessary adjuncts to dietetic and tonic treatment. Should the bones have already some degree of curvature further yielding is prevented by placing the legs in restraining splints, which if carried beyond the feet will efficiently prevent any attempt at walking or standing when the mother's back is turned.

§ 447. II. Under **chronic osteitis** and **periostitis** are included a number of tuberculous, syphilitic and other conditions leading to caries, necrosis and other anatomical changes in the bone. Osteitis and periostitis may be dealt with together, for although the disease may start in the bone or the periosteum it soon spreads to the other.

The *symptoms* of osteitis and periostitis may have come on with acute pain, redness and swelling; but more frequently they come on insidiously with hardening, thickening, or enlargement of the bone. These symptoms may be followed by suppuration (caries) or death of a portion of the bone (necrosis) with signs of abscess formation.

Causes and their differentiation. (1) *Traumatism* alone without sepsis or toxæmia of some kind is a rare cause of chronic periostitis or osteitis. Traumatism is recognised by its history, and by the fact that only one bone is affected.

(2) The favourite seat of *tubercle* is the epiphysis, where it induces a chronic epiphysitis, especially in the neighbourhood of the hip or knee. Sometimes it gives rise to osteitis, and when this occurs in the fingers it results in a characteristic thickening of the phalanges known as "strumous dactylitis." In any position it may go on to caries or necrosis. Tuberculous affection of the bones is recognised by (i.) the youthful age of the patient; (ii.) a tuberculous history; (iii.) by the characteristic intermitting pyrexia; (iv.) signs of tubercle in the lungs and elsewhere; (v.) by

the chronicity of the process; and (vi.) the frequent limitation to one bone.

(3) *Syphilitic* affections of the bones are very common both in the acquired and the hereditary disease. a. *Acquired syphilis* may take the form of a chronic diffuse or localised periostitis (nodes), or, on the other hand, a diffuse or a gummatous (localised) osteitis. It is recognised by (i.) the characteristic flying pains in the limbs; (ii.) the nocturnal pains in the bones which are such a frequent manifestation of syphilis: and (iii.) other evidences of syphilis. b. *Hereditary syphilis* may give rise in childhood and early life to the same lesions as the acquired disease. In infancy (in addition to the foregoing) chronic suppurative osteochondritis (chronic epiphysitis) is apt to arise and to be mistaken for rickets. In this condition one or several bones may be affected, but it never presents the same symmetry as rickets. The deformities resulting from hereditary lesions (Table I., p. 34) and the physiognomy (p. 26) are very often characteristic—the bosses on the frontal and parietal bones (Parrot's nodes), the depressed bridge of the nose, scars about the angle of the mouth, Hutchinson's teeth (Fig. 3, p. 25), and perhaps keratitis.

(4) Rheumatism and gout may give rise to chronic periosteal thickening, or periosteal nodes.

For the adequate *treatment* of most of these different conditions, rest and surgical aid are necessary. The treatment of the tuberculous, syphilitic, rheumatic and gouty diatheses, has already been dealt with.

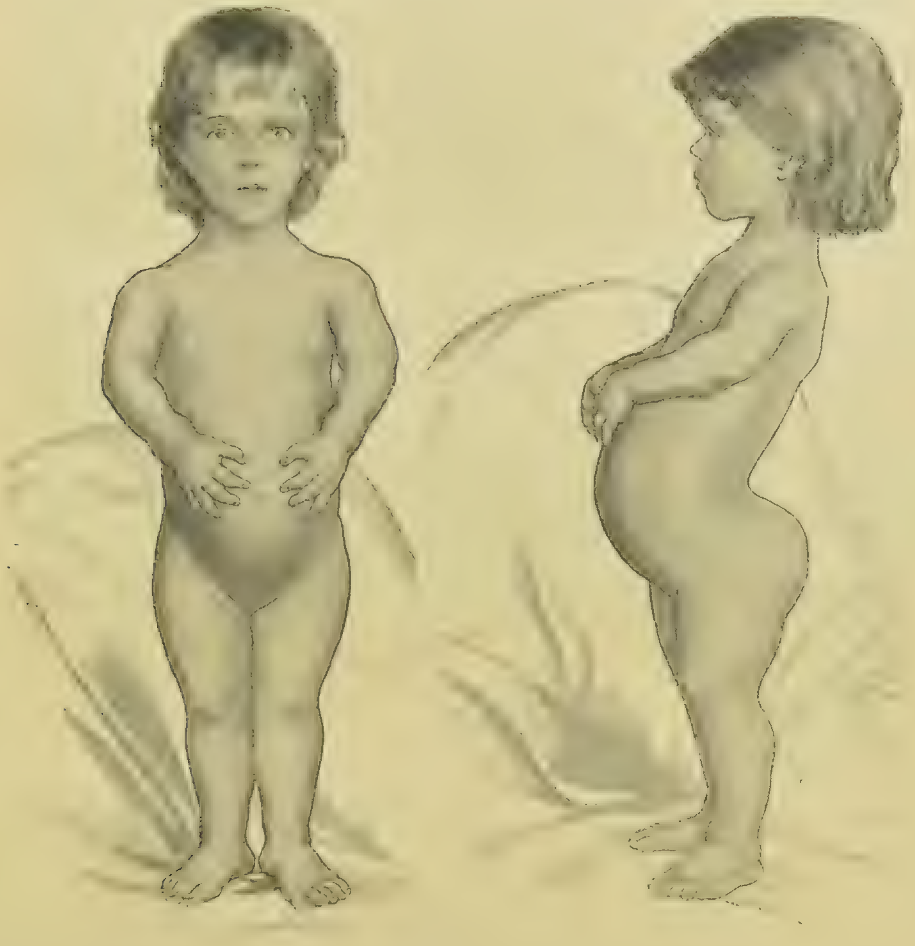
III. **Tumours of Bone** may commence with pain, tenderness, and swelling like chronic periostitis. The chief *innocent* tumours are EXOSTOSES, which may occur on almost any bone, and ENCHONDROMATA, which are commonest on the metacarpals and phalanges. Both are usually multiple. The *malignant* tumours are either SARCOMA (especially myeloid sarcoma) or CARCINOMA. In both the swelling of the bone is more rapid and reaches a greater degree than in any of the other causes of swelling above mentioned, and as a rule they are limited, at any rate at first, to a single bone. Sarcoma of bone may occur at any age; carcinoma is limited to advanced life and accompanied by the usual cachexia.

§ 448. There are six rare forms of chronic bone disease which need to be mentioned.

IV. **Acromegaly** is a rare disease first described by Dr. Pierre Marie, leading to enlargement of the skeleton. The patients generally apply for treatment for some other malady, though sometimes they apply on account of the awkwardness of their movements, and sometimes they complain of obscure pains in the limbs. The aspect is very characteristic. The bones and other tissues of the hands and feet become markedly elongated and hypertrophied, though the growth is so gradual as to escape the patient's notice. The cranium is increased, but not so much in proportion as the face, which is egg-shaped, the lower jaw representing the large end of the egg. The lower jaw especially is enlarged and may project beyond the upper jaw. The nose and the cranium are also enlarged, while the thickening of the soft parts causes hypertrophy of the ears, eyelids, nostrils, and tongue. Later in the disease there may be a similar enlargement of the bones of the limbs and the thorax, and kyphosis of the spine. Sometimes there is temporal hemianopsia, with gradual optic atrophy.

Diagnosis. Myxœdema resembles acromegaly, but it is known by its round or "moon-shaped" face, the dry skin, and the absence of all bony enlargement or muscular weakness. Pulmonary osteo-arthropathy (see below).

Acromegaly occurs rather more frequently in women, generally beginning about the 25th year. Changes in the pituitary body, either hypertrophy or tumour, have been found in all the fatal cases. It has been suggested that gigantism and acromegaly are one and the same disease, and that both are due to disordered function of the pituitary gland, and it is worth noting that in the skulls of certain giants the sella turcica has been found to be considerably enlarged. In some cases there have also been changes in the thymus or the thyroid glands, and mediastinal dulness has been made out during life in the position of an enlarged thymus.



Figs. 127 and 128.—A case of ACHONDROPLASIA (p. 816) exhibited by Mr. W. Turner at the Clinical Society of London (Trans. Clin. Soc. Lond., vol. xxxii., 1899).

Treatment. Acromegaly runs a very prolonged course of many years and no known treatment seems to affect it. Extracts of pituitary gland and thyroid have been tried. The patient generally dies of some intercurrent malady.

V. **Achondroplasia** (syn. Fœtal Rickets; Chondrodystrophia fœtalis) is a rare condition of infancy leading to dwarfism and generalised deformity, which until a few years ago was probably confused with cretinism on the one hand or with the deformity resulting from rickets on the other. Figs. 127 and 128 (p. 815) represent a case exhibited at the Clinical Society of London by Mr. W. Turner, to whom the author is indebted for the photographs. There is a generalised symmetrical shortening of the diaphyses (producing characteristic shortening of the limbs) with considerable thickening of the epiphyses (producing enlargement of the articulations), due to hyperplasia of the cartilaginous ends of the bones. Consequently the stature is stunted, the fingers and toes taper and are abducted from one another, the cranium is large, the face small, and the bridge of the nose depressed. The disease is congenital and dates from birth. The mental deficiency, facial aspect, and the changes in the hair and skin characteristic of cretinism are absent, and cases do not exhibit the constitutional symptoms or characteristic changes of rickets in the skull.

VI. **Pulmonary Osteo-arthropathy** is a chronic osseous hyperplasia sometimes associated with chronic pulmonary disorders. There is enlargement of the hands and feet and of the lower ends of the long bones of the legs and forearms, but the face and head are not enlarged. The nails are curved over the enlarged terminal phalanges, "filbert nails."

VII. **Osteitis deformans** (syn. Paget's disease) is a somewhat rare disease coming on after middle life, mostly in males, and consisting of a very chronic enlargement of the bones, both in diameter and in length. The histological change is a rarefying osteitis with enlargement of the Haversian spaces. It affects the cranium (not the face), spine, limb bones, and clavicle. It becomes manifest to the patient by the fact that he frequently has to change the size of his hat. Sometimes rheumatic pains in the bones are complained of.

VIII. **Mollities Ossium** (syn. Osteomalacia) is a progressive disorder of the bony system, due to gradual decalcification and weakening of the skeleton which results in considerable deformities and contortions, owing mainly to muscular action, occurring in women (91 per cent., Arthur Durham) between 25 and 35 years of age, mostly after pregnancy. The early symptoms consist of wandering pains in the limbs and trunk, worse at night, with weakness of the limbs. In the course of a few months there is bending of the bones; spontaneous fractures and distortions may occur. The stature is diminished from the involvement of the spine. Death usually occurs from respiratory complications owing to fracture of the ribs.

IX. **Leontiasis Ossea** is the term given to a rare condition in which there are symmetrical hyperostoses of the facial bones and skull, which encroach upon the cranial cavity and so may lead to death.

CHAPTER XVIII.

THE SKIN.

The skin is subject to the same diseases as other epithelial structures, but the circumstance which strikes the thinking student is that, although the various diseases have, as it were, written their own characters on the surface of the body in full view of the observer, we nevertheless know less really about the pathology of morbid processes which occur in the integument than we do about those which take place in most of the other parts of the body.

Another circumstance which increases the mystery surrounding skin disease is the immense number of names adopted by dermatologists for the same or but slightly different diseases. It has not been possible to give all the synonyms for the various recognised types of disease—even were I competent to do so—in the following pages, but a glance at the disease now usually known as dermatitis herpetiformis will give some idea of the mysteries and difficulties of this nomenclatural maze. It would almost seem as though a dermatologist was compelled to rename some old disease or to rediscover some "new" disease in order to justify his pretensions to the title.

PART A. SYMPTOMATOLOGY.

The cardinal symptom of skin affections consists of an ERUPTION with or without SUBJECTIVE SYMPTOMS. The subjective symptoms of skin diseases are of relatively less importance for diagnostic purposes, because the morbid process itself is before us. There is, however, one subjective symptom which attends a great many skin diseases, namely, PRURITUS (itching). Anæsthesia and other disorders of sensibility will be dealt with among diseases of the nervous system.

§ **449. Pruritus** is the Latin word for itching, and that is the sense in which it is used here. It may be remarked in passing that prurigo is a disease in which itching is the leading and most invariable sign, although papules and scratch-marks are generally, and an urticarial element sometimes present.

There are 3 groups of *causes* of itching :—

a. Pruritus may be *secondary* to some visible skin disease, and in that case the itching is localised to the neighbourhood of the eruption. Some eruptions are invariably attended by itching, such as urticaria, eczema, and most acute conditions which progress rapidly. Other diseases are almost invariably unattended by itching, such as syphilis, psoriasis, and most chronic conditions which evolve their course slowly.

b. Various *local conditions* may produce more or less localised itching :—1. *Discharges* or secretions from nasal, buccal, or anal orifices, *e.g.*, pruritus ani, pruritus scroti, pruritus pudendi. In many of these cases there is also a certain amount of localised eczema, which is possibly also the result of the discharge or sweat. 2. A *rough garment* such as a new flannel shirt or certain dyed articles may produce intolerable itching in delicate skins. 3. Various *parasites* give rise to pruritus. With *scabies* there is also a characteristic eruption localised chiefly to the flexures of the joints. In *phtheiriasis* (due to pediculi corporis) the eruption is generalised, though most intense across the shoulders ; the *flea*, the *harvest-bug*, *pediculus pubis* and other parasites cause intense itching.

c. With *idiopathic* or internal causes the itching is generalised, and may or may not be accompanied by a certain amount of generalised eruption of papules (see prurigo, § 463). Among the causes may be mentioned gout, certain articles of food (*e.g.*, shell-fish, eggs, cheese, excess or deficiency of salt), jaundice, digestive disorders, lithæmia, diabetes, kidney disease, nervous irritability, constipation, and old age. The pathological condition on which all of these depend is in the author's view[1] some blood change. The *treatment* of pruritus is given under prurigo.

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[1] *The Lancet*, Aug. 1, 1896, p. 300.

PART B. PHYSICAL EXAMINATION.

The APPARATUS required for the investigation of skin diseases is simple, and consists of a good lens some 3 inches in diameter, a microscope with accessories, and the means of histological examination. A pair of flat forceps is useful for removing scales, hairs, or parasites. A flat glass slide may be used or the skin may be stretched to ascertain if the spots disappear on pressure.

HISTOLOGICAL EXAMINATION is of great use in many cases, and frequently enables one to diagnose a lesion with certainty: a small piece of the diseased skin can be removed without causing appreciable pain if the part be first frozen by ethyl chloride. As the skin freezes pinch up the fold required with the fingers so that it retains its shape, then take hold of it with a forceps and cut with curved scissors a tiny piece (including all the layers of the skin). which can be put into alcohol. then celloidin, and mounted next day.

§ 450. The points to investigate in any given case of skin eruption are—I. The size and appearance of the prevailing elements; II. What it feels like and whether it disappears under pressure; III. The position and symmetry of the eruption; IV. Subjective symptoms; V. The duration and evolution of the eruption; and VI., its etiology.

I. The **character and size of the prevailing elements.** The spots are *never all quite alike*, being modified by the age of each spot, the locality affected, and the conditions to which it has been subjected (*e.g.*, scratching or pressure). It is therefore of the highest importance to *examine every part* of the eruption. Patients may object to undress and the physician may grudge the time, but these considerations should never be allowed to weigh. The most convenient clinical classification of skin diseases is based upon the nature of the elementary lesions, a list of which is given below.

§ 451. The principal **elementary lesions** which appear on the skin are as follows. There are 3 varieties of primary lesion, and 3 which arise secondarily to these.

1. A *macule* (or macula) is a spot of congestion; *roseola* is a generalised eruption of macules; *erythema* is a larger area of congestion with fading edges. A *wheal* is a spot of congestion accompanied by slight exudation beneath the skin; it is also called *urtica*, and a generalised eruption of wheals is

called *urticaria* or "nettle-rash," because they resemble nettle stings.

2. A *papule* (or pimple) is a small solid elevation of skin, conical, round-topped, or flat. A *lenticular* papule is a large flat-topped papule. A tubercle or *nodule* as it may be better called, to avoid confusion with the lesion of tuberculosis, is a localised cellular infiltration of the skin.

3. A *vesicle* is a collection of serous fluid beneath the cuticle. A *bulla* is a large vesicle. A *pustule* is a collection of purulent fluid beneath the cuticle.

The SECONDARY lesions are:—

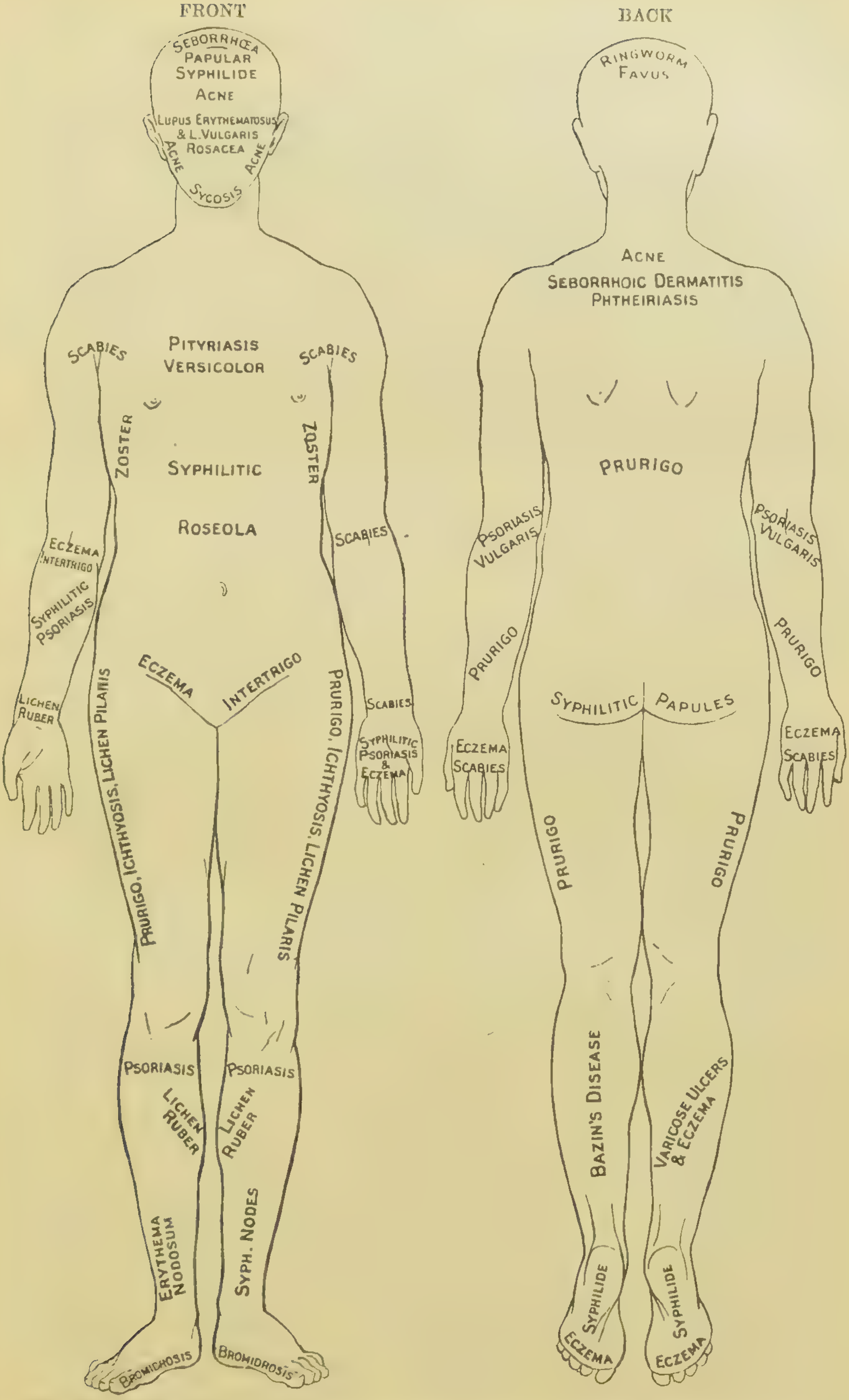
1. A *scale* or squame is the exfoliation of cuticle which occurs after a congestion or inflammation of the skin, or it may be the product of pathological processes special to the skin, such as cornification, or hyperkeratosis. In a sense a scale may be a primary lesion.

2. A *crust* or scab is dried serum or pus.

3. *Fissures*, ulcers, cracks, excoriations are breaches of the surface. *Cicatrices* or scars may result from these when a sufficient extent or depth of skin is involved.

PIGMENTARY ALTERATIONS are known as *chloasma* when there is a broad streak of excessive pigment; leucoderma, when there is an area of skin devoid of normal pigment; melanoderma, when there is an area of increased pigmentation. *Ephelis* is a freckle. *Nævus* is a mole or birthmark, either pigmented, hairy, or vascular. A dilatation of the superficial vessels of the skin is known as *telangiectasis*. *Petechiæ* are small spots of hæmorrhage into the skin. *Ecchymoses* are larger patches of extravasated blood which go through the changes of colour characteristic of a bruise. A *comedo* or "blackhead" is a little black plug of inspissated serum blocking the orifice of a sebaceous gland.

The fundamental **histological changes** of the skin are congestion (hyperæmia) with or without exudation, inflammation, and infiltration. If the lesion consists of *congestion*, such as roseola, or urticaria, or simple *inflammation* without infiltration, such as eczema, it disappears on pressure. If, on the other hand, there be definite *infiltration* or neoplastic deposit, as in lupus and syphilis, or if there be *hæmorrhage* into the skin, the colour does not disappear when the skin is pressed by the finger or a glass slide, or stretched. This is a point of much significance in the diagnosis of skin diseases. The secondary consequences of inflammation in the skin are as elsewhere three in number. If the inflammation does not undergo resolution, there may be (1) *suppuration* leading to the formation of pustules, ulcers, etc.; (2) *necrosis*, as in the centre of boils and carbuncles; or (3) *[illegible]*, as in the case of the various scars, hypertrophies, or scleroderma. In addition to the primary lesions just referred to—congestion, inflammation, and infiltration and their consequences—which occur in the skin as elsewhere, there are at least 4 processes special to



Figs. 129 and 130.—Diagram showing the parts most frequently affected by certain eruptions.

the skin. 1. *Hyperkeratosis* is an increased deposit of kerato-hyaline material leading to an increased cornification of the surface cells of the epidermis, and a scaliness of the surface as in pityriasis and psoriasis. 2. *Parakeratosis* is the irregular or deficient cornification which occurs for instance in eczema. Here the prickle cells instead of going through the regular process of cornification by the deposit of kerato-hyaline granules in their interior, and their gradual conversion into dry horny non-nuclear cells, remain moist and succulent (though dry on their exterior), and retain their nuclei. They adhere to one another, being moister, and are shed in masses of crusts and scales instead of being shed singly and imperceptibly. 3. *Acanthosis* is a term applied to the increased proliferation of the prickle cells by increased mitosis (karyokinesis), resulting in an increased thickness in the epithelial layers of the cuticle. 2 and 3 are found in all kinds of eczema, 2 chiefly in dry eczema; 3 is met with in moist eczema.

II. What does the **eruption feel like,** and does it **disappear on pressure?** Infiltrating lesions feel hard, and do not entirely disappear on pressure, as is evident from the histological characters (*v. supra*). A faint purpuric eruption may thus be diagnosed from an erythema.

III. The **position** and **symmetry of the eruption** is important for purposes of diagnosis, and on these grounds also it is essential to examine the whole of the eruption. Many diseases may be recognised by the position in which the elements predominate, and Figs. 129 and 130 will aid the student to remember the parts most frequently affected by certain eruptions. Some diseases are always more or less generalised, *e.g.*, urticaria and the exanthemata, and this generalised distribution usually indicates, as in the eruption also of secondary syphilis, a toxæmic or idiopathic cause. Others, while sometimes affecting the whole body, have a preference for certain parts, *e.g.*, psoriasis for the knees and elbows, seborrhœic dermatitis for the head and shoulders. Various words are used to describe the distribution, thus, *punctate* when the eruption is dotted about, *discrete* when the elements are separate, *confluent* when they run together, *gyrate* or crescentic when they are arranged in wavy lines or segments of circles, *circinate* or annular when they are in circles, *corymbose* when grouped into clusters.

Any **symmetry** of arrangement on the two sides of the body should be carefully observed, though its significance must not be overrated. It may indicate that some constitutional or blood change is in operation as in the earlier eruptions of syphilis. Symmetry may also indicate that some nervous or neuro-vascular cause is in operation, as in certain erythematous eruptions. But perhaps the commonest cause of symmetry is the fact that both of the parts involved are exposed to the same extraneous conditions.

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as in eczema of both hands due to washing in strong solutions of soda.

IV. The presence of **subjective symptoms** must be inquired into, such as itching, burning, smarting, etc. Syphilitic eruptions do not usually itch, a feature which helps to distinguish them from the corresponding non-syphilitic rash. A large proportion of skin diseases are *unattended by constitutional symptoms*, if we except tuberculous and syphilitic eruptions, and the eruptive fevers.

V. The **duration of the eruption** and the history of its **evolution** must also be investigated. The rate at which a disease has developed is a most important aid to diagnosis. For instance, lupus vulgaris will not produce so extensive a lesion in the course of years as a facial syphilide which resembles it will produce in the course of weeks or months. It must be remembered also that during its progress, a skin disease may alter its appearance considerably; a lesion which starts as a papule may become a vesicle and then a pustule, as in small-pox.

VI. The **etiology** must also be inquired into. First as regards PREDISPOSING causes: 1. The *age* of the patient. Lupus vulgaris nearly always starts in early life, but lupus erythematosus rarely starts until middle life. 2. *Sex* does not aid us much in the diagnosis, though lupus erythematosus is more frequent in the female. 3. *Heredity* is not a potent factor in skin disease, though ichthyosis, psoriasis, cancer, and albinism have been traced in families. 4. The *occupation* of a patient may result in certain skin diseases by a want of cleanliness, or may cause various forms of eczema of the hands (grocer's itch, baker's itch, etc.).

Among the EXCITING causes—1. *Traumatic conditions* frequently produce a lesion which is indistinguishable from eczema, and, as already noted, friction and scratching may modify the characters of an eruption very considerably. 2. *Parasites* produce eruptions which have characters special to them. 3. *Vegetable organisms*, fungi, on the surface of the body cause ringworm, favus, and other diseases. 4. *Bacteria* introduced *into the body* are the cause of the exanthemata. 5. *Gout* and other toxæmic conditions are the causes of some eruptions, *e.g.*, urticaria, erythema, prurigo, etc. 6. Many *drugs* are attended by characteristic eruptions (§ 456). 7. *Diseases of the internal organs* may produce eruptions, especially digestive disturbances (urticaria), disease of the peripheral nerves and their ganglia (herpes and glossy skin and other trophic changes), acute and chronic Bright's disease.

PART C. DIAGNOSIS, PROGNOSIS, AND TREATMENT OF SKIN DISEASES.

§ 452. Routine Procedure and **Classification.** The LEADING SYMPTOM is generally before our eyes. The HISTORY, DURATION, and MODE OF EVOLUTION can be inquired into while the patient undresses. Then we proceed to the PHYSICAL EXAMINATION as described in Part B.

If the eruption is QUITE DRY and consists of wheals, macules or erythema, papules, or scales, turn first to	§ 453
If the eruption is MOIST or consists of serous exudation, vesicles or crusts, turn first to	§ 477
If the eruption consists of *pustules*, turn first to	§ 483
If it is *multiform*	§ 488
If it is *nodular*	§ 489
If there is *ulceration*	§ 491
If there are *warts* or *excrescences*	§ 492
If there are *scars* or *atrophies*	§ 493
If there are *vascular* or *pigmentary* alterations	§ 494
If there is disorder of the *sweat*	§ 496
If the *hair* or scalp is affected	§ 498

GROUP I. ERUPTIONS USUALLY DRY — *wheals; macules or erythema; papules; and scaly eruptions.*

a. Wheals.

§ **453**. **Urticaria** ("nettle-rash") is a generalised eruption which consists of wheals, of more or less evanescent character, rarely lasting more than a few hours. Patients come complaining of the *history* of such an eruption accompanied by intolerable itching; and sometimes although there are no wheals visible these can readily be produced by drawing a point across the skin (dermatographia or urticaria factitia). This latter condition may very frequently be found in association with the exanthems and erythemas. It is also present in states of cerebral congestion (meningitis) and constitutes the tâche cérébrale of Trousseau.

Etiology. Some people, especially neurotics, develop urticaria upon the slightest cause, for instance, on meeting a stranger, but the commonest determining cause is the eating of shell fish, or unwholesome tinned food, or some intestinal toxin or gastro-intestinal derangement.

Varieties. 1. There is an acute and chronic form of the affection; the first named consists of a transient attack lasting a few hours or days; in the chronic or more properly the *recurrent* form (urticaria perstans) there are constantly recurring attacks. 2. Urticaria papulosa, see § 463, and 3. Urticaria pigmentosa (§ 494). 4. In giant urticaria or Quincke's disease the eruption is more persistent and consists of circular white œdematous spots or lumps in the skin. 5. Angio-neurotic œdema consists of still larger lumps affecting the loose cellular tissue. 6. Urticaria bullosa is a rare variety with vesicles or bullæ, met with chiefly in children.

Prognosis and *Treatment.* The disease as usually met with is essentially transient, and a brisk saline purge is all that is necessary. Relapsing cases are, however, difficult to cope with, and in these strict attention to the diet and the digestive organs is called for. The remarks under Erythema Multiforme concerning the coagulability of the blood and the treatment by calcium chloride apply in my experience equally here. After any digestive defects have been corrected, it is wonderful how efficacious 20 grain doses of calcium chloride thrice daily are in some cases, though in others the eruption reappears on ceasing the drug.

b. Eruptions which usually consist of Macules or Erythema.

Generalised.	*Localised.*
I. Exanthemata.	I. Rosacea.
II. Roseola (simplex and syphilitica).	II. Lupus erythematosus.
III. Erythema scarlatinoides.	III. Erythema nodosum.
IV. Drug eruptions.	IV. Erythematous eczema, X-ray dermatitis, Erythema paratrimma, E. faciei, E. traumaticum, E. caloricum, E. Pernio, and other varieties of E. multiforme; Macular Leprosy, and Pellagra.
V. Erythema multiforme.	

The early stages of eczema and of other eruptions to be mentioned hereafter may take the form of an erythema.

I. The **Exanthemata** or eruptive fevers are fully described in Chapter XV. where they form Group I. of the acute specific fevers.

§ 454. II. **Roseola** is a term employed to designate a generalised eruption consisting of patches of congestion, more or less marginated, varying in size from a pin's head to a lentil. Two varieties are described.

R. Simplex may resemble measles, and indeed its chief importance is in connection with the diagnosis from this disease (*q.v.*). It gives rise to a considerable amount of itching and irritation, with usually a slight degree of constitutional and gastro-intestinal disturbance. It may occur in childhood under the same conditions as urticaria, and is therefore predisposed to by gastric disturbance. The occurrence of such an eruption when small-pox is prevalent should make one suspect the initial eruptions of that disease. It is one of the commonest rashes associated with vaccination. Drugs, such as copaiba, may cause it. *Treatment* consists in administering an aperient, salines and diuretics, and in correcting any concurrent digestive disorder.

Roseola Syphilitica is the earliest of the syphilitic skin eruptions, occurring 3 to 6 weeks after infection. It appears upon the trunk, chiefly its anterior aspect, the chest, the flexures of the limbs and the palms and soles, as rosy or *dusky* red macules, disappearing on pressure, rounded, oval or irregular in shape with fading edges, varying in size from a pea to a shilling. A degree of pigmentation may be left behind. Sometimes the eruption is so faint that it is overlooked. It becomes better marked after a bath or when the skin is exposed to cold. It may last from a few days to a few weeks. It is *diagnosed* by the history and other signs of syphilis. *Non-syphilitic roseola* undergoes rapid changes in size and shape; *pityriasis versicolor* can be scraped off, and is fawn-coloured; *seborrhœic eczema* develops greasy scales on the surface, and forms spots of irregular size and outline.

§ **455.** III. **Erythema Scarlatinoides,** as its name implies, consists of a widespread rash, resembling scarlet fever, preceded and accompanied by fever and constitutional disturbance, and followed by desquamation. So-called "surgical scarlatina" is probably identical with this condition. The chief causes are septicæmia, intestinal disorders, enemata of soap or other substances, the ingestion of ptomaines and other toxins, certain drugs (see below), rheumatism, gonorrhœa, and sewer-gas. The *diagnosis* from scarlet fever is difficult only in severe cases. In erythema there is less constitutional disturbance, no strawberry tongue, and there is a tendency to relapse.

§ **456.** IV. **Drug Eruptions.** An idiosyncrasy with regard to certain drugs, whether taken by mouth or applied externally, is shown by some individuals and is manifested by the appearance of a rash, which disappears on the withdrawal of the drug. Two things will be noticed in the list below: (1) that by far the commonest eruptions are erythema and its congeners; and (2) that the iodides and bromides are among the commonest drugs to produce eruptions; they may even produce a frambœsial eruption resembling gumma. The chief eruptions produced by the internal administration of drugs are as follows.

Papulo-pustules:—bromide and iodide of potassium, occasionally sulphide of calcium, antimony and arsenic (from both internal and external application).

Erythema:—antipyrin, antitoxins, copaiba, cubebs, turpentine, belladonna, atropine, quinine, salicylic acid and sod. salicyl, boracic acid; iodoform and carbolic acid by absorption from wound dressings. And in the author's view certain toxins absorbed with milk.

Urticaria:—quinine, copaiba, turpentine, valerian, santonin, soda salicylate, benzoic, salicylic and tannic acids.

Erysipelatoid (erythema with infiltration or œdema of the skin):—bromide and iodide of potassium, quinine, iodoform, boracic and carbolic acids (aconite, oil of cade, chrysarobin and carbolic acid applied externally).

Herpes:—arsenic.

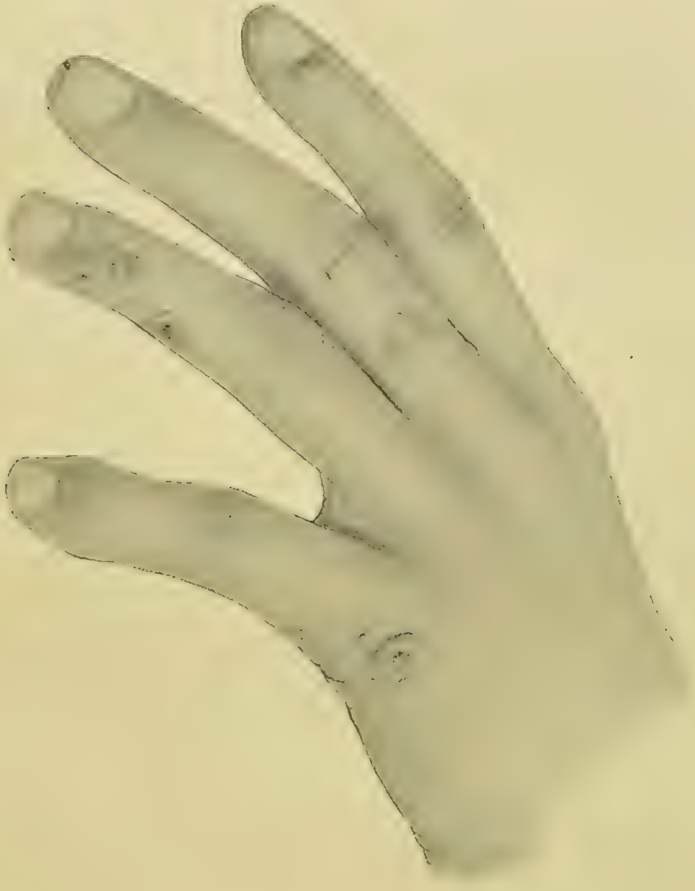

Fig. 131.—Erythema Iris on the hand of a single woman 23 years of age.

Purpura:—iodide of potassium, chlorate of potash, chloral hydrate, chloroform, copaiba.

Pigmentation:—silver nitrate, arsenic.

Epidermic thickening:—arsenic and borax.

§ 457. V. **Erythema Multiforme** is an affection characterised by erythematous efflorescences, varying in form and size, localised usually upon the backs of the hands and forearms, dorsum of feet and legs, and sometimes on the face, neck, and the trunk, and accompanied usually by lassitude and ill-health. The lesions vary in size from a lentil to the palm of the hand. They belong to the *congestive* form of eruption, being slightly raised, with fading edges. The centre is the highest part, is usually livid.

and sometimes hæmorrhagic. There is usually a feeling of burning or formication. Many varieties have been described, such as *e. gyrata*, when the blotches fade in the centre, coalesce with neighbouring ones, and form wavy or gyrate lines. *E. iris* (herpes iris, herpes circinatus) is a form in which a vesicle forms in the centre, with concentric rings of purple and white and red around it (Fig. 131). In *E. bullosum* bullæ form. *E. nodosum* is described in § 460. Erythema is known from urticaria by its deep red coloration, by its more localised distribution, the larger size and more permanent character of the lesions, less itching and more marked constitutional symptoms. Young people and males are more prone to the affection. It is commoner in the spring and autumn. The *course* of the disease varies, but each erythematous patch lasts 8 to 10 days, and they continue to appear for 2 to 6 weeks. Each may leave temporary brown pigmentation, and desquamation may occur as they fade.

Treatment. That the disease is due to some alteration of the blood is shown by the marked effect which certain remedies have upon the condition, especially quinine, and, as Dr. A. E. Wright and the author have shown, calcium chloride.[1] This alteration appears to consist of a diminished coagulability of the blood, such as occurs when calcium salts are deficient. Quinine should be given in 4 or 5 grain doses, t.i.d. and gradually diminished, or gr. xx. calc. chlor. If given after meals these doses do not impair the digestion. The diet and digestive system require careful regulation.

Erythema of more or less LOCALISED *distribution.*

§ 458. I. **Rosacea** (syn. Acne Rosacea; A. Erythematosa; Gutta Rosacea Kupeferrosa) presents three stages: (1) simple congestion or erythema attacking the nose and adjacent parts of the cheek, often worse after meals. (2) In the next stage dilated vessels (telangiectasis) appear upon the surface, and after some time, inflamed sebaceous follicles (acne papules and pustules) appear. (3) The third stage involves considerable hypertrophy of the connective tissue elements of the skin, leading to the formation of nodules (rhinophyma) which are sometimes of great size. Rosacea runs a prolonged course: the first stage alone may extend over many years.

The *diagnosis* is not difficult, except in its early stage, when the erythema may be mistaken for lupus erythematosus and other kinds of erythema of the face (§ 461). The latter, however, is recognised by slight scaliness; and a lens reveals the presence of fine cicatrices.

The *causation* of the disease is not understood. It affects both sexes, but in the female sex is particularly apt to start at the evolution and involution of sexual life, *i.e.*, when the vaso-motor system is particularly irritable. Among males, it is specially apt to affect cabmen, coachmen, mariners and others who are exposed to the weather. Drink is a frequent and potent cause but the disease may arise in the most abstemious persons.

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[1] *Lancet*, Aug. 1, 1896.

Much of the *treatment* of acne vulgaris (§ 462) is available for rosacea. The cause if possible should be removed. In the first stage stomachics, alkaline carbonates and other internal remedies are indicated. Several cases under my care have benefited by large doses of calcium chloride. Lotio calamine and soothing remedies should be applied. In the later stages, diligent application of phenol at intervals, and for rhinophyma scarification, will undoubtedly ameliorate even very bad cases.

§ 459. II. **Lupus Erythematosus** (syn. ul-erythema: seborrhœa congestiva of Hebra) is the most chronic of the erythemata. The eruption has a spreading erythematous border, which as it spreads leaves a very thin permanent scar in the centre. In the first stage the disease begins with one or more small, red, slightly raised spots covered with a thin adherent scale. By an increase in number, the little patches form, in the course of many months, an irregular bluish red area with thin cicatricial centre and erythematous margin covered with scales and sometimes with crusts. In another variety (seborrhœa congestiva of Hebra) there is a marginated erythema with numerous black specks, or large gaping openings of the sebaceous glands: the central part of the skin appearing depressed and covered with adherent dry scales, interspersed with venules. The favourite seats of the eruption are the cheeks and bridge of the nose (butterfly distribution); then other parts of the face and forehead, the lips, ears, scalp (where the scar leaves permanent bald patches) the extensor surfaces of the hands, fingers and toes (l. pernio), and more rarely on other parts of the body. The patches are generally symmetrical.

Etiology. The disease is much more frequent in women than men and very rarely occurs under 20, a most important circumstance in the diagnosis from lupus vulgaris, which invariably appears before or during adolescence. The resemblance of l. erythematosus and l. vulgaris and the family history of tuberculosis, according to some observers, have suggested the idea the former is also connected with the tubercle bacillus, but this has never been found in the lesions. The *diagnosis* between these diseases is given in tabular form (§ 489). *Prognosis.* L. erythematosus extends over 10 or 20 years; always terminates in cicatricial changes in the skin, and permanent baldness of a hairy part. Beyond the disfigurement the disease is not serious, and is unattended by constitutional symptoms.

Treatment. In the early stage we must employ soothing remedies which act superficially (*vide* acute eczema). If these fail, stimulating ointments and caustics may be necessary, such as salicylic and carbolic ointment or salicylic collodion, or plaster mull. Painting with carbolic or carbol-camphor, or sulphur pastes, etc., give rise to a reactionary inflammation and swelling which runs its course in a few days, then results in considerable improvement. Linear scarification, X-rays, high frequency and Finsen treatment have also given satisfactory results. Internally, quinine, salicylic acid and intestinal disinfectant treatment have given excellent results.

§ **460.** III. **Erythema nodosum** is an eruption with an acute onset, consisting of erythematous lumps about the size of a pigeon's egg, occurring most frequently over both shins. The patches are round, oval, raised, non-marginated, painful and tender. The centre is most deeply coloured, whence the purplish tint gradually fades away to the margins. There is usually some malaise and elevation of temperature: sometimes pain in the joints and other rheumatic symptoms. Each nodule lasts one to two weeks and successive crops may continue for a month or two. Patients are usually young women, and there is often a disturbance of the catamenia. The rheumatic diathesis is often present. The condition is known from other forms of erythema by the position of the lesion and the acute pain and tenderness. In periostitis the lesion is usually single. The disease usually runs a benign course to spontaneous recovery in a month or two. The *treatment* consists in the administration of salicylates, saline aperients, and after the acute symptoms have subsided, iron and quinine internally. Lead and opium lotion applied locally allays the pain.

§ **461.** IV. Certain specially-named forms of erythema may be mentioned.

Erythematous Eczema. Eczema is usually a vesicular disease, but there is an erythematous variety which may run its course without presenting any vesicles. The surface of the skin is red, dry and rough, with slight scaling. It frequently attacks the face, when the eyes may be almost closed, and is attended by burning and itching. For treatment, see eczema, § 477.

X-ray Dermatitis is a form of erythematous eczema. The Röntgen rays produce at first slight erythema, which may go on to vesiculation and fine scaly exfoliation, while frequent and prolonged exposures lead further to pigmentation, loss of hair, and considerable pain. Serious ulceration has occasionally supervened. Treatment is prevention by shorter exposures and longer intervals, and on the part of those engaged in X-ray work greater protection by wearing lead foil or other impervious matter.

Bedsores (E. paratrimma) are due to pressure over prominent parts, such as the sacrum, trochanters, heels, or ankles of the bedridden, or to the pressure of a badly-adjusted splint. A local patch of erythema appears, followed by abrasion of the skin, developing into an eczematous condition. If the cause continues sloughs form. They are due to three causes :—pressure or irritation from rubbing, perspiration and excretions in cases of incontinence, the lowered vitality of the sick and aged. In certain nerve diseases, especially myelitis, the sloughs form so rapidly in spite of all precautions that the condition is attributed to a trophic neurosis. Extreme fœtor attends the decomposition of the slough, and septicæmia may set in with fatal result.

Treatment. Good nursing can prevent bedsores. Three principles should be kept in mind :—cleanliness, dryness, and relief of pressure. (i.) The parts should be carefully cleansed night and morning, and the draw-sheet pulled through immediately it becomes soiled. (ii.) After washing, the skin should be thoroughly dried by rubbing over a little methylated spirit or brandy, and well powdered. (iii.) Relief of pressure is obtained by a water-bed, ring pads, and by frequently turning the patient from side to side. If an ulcer or slough forms these measures should be combined with the plentiful use of antiseptic lotions, and occasionally a charcoal poultice to relieve the pain and promote healing. The diet should be liberal and stimulating, especially in the aged.

Erythema faciei vel *e. fugax* is a flushing of the face which occurs chiefly in association with dyspepsia. It may form the first stage of rosacea. *E. traumaticum* develops on any part subject to long-continued pressure, *e.g.*, the garters and tight waist-bands. *E. læve* is the erythema found on the legs of dropsical persons. *E. caloricum* is the erythema which appears on the face from exposure to the sun and wind. *E. intertrigo* is found in parts which are opposed, such as the thighs and armpits, in infants, corpulent people, or those who perspire much. It may pass on to eczema. *E. pernio* (syns. dermatitis congelationis, frostbite, chilblain) may be defined as a painful inflammatory condition of the skin of the fingers, toes, heels, or other portions of the feet, hands, or limbs, caused by exposure to cold, and attended with itching and tenderness, sometimes by vesication, ulceration, or gangrene. In the form of the so-called chilblain it is prone to occur in those whose circulation is poor, and constitution feeble. Thus children and old people frequently suffer from this complaint during successive winters. *Treatment* consists in local applications of stimulating liniments, such as iodine and camphor, and internally iron and strychnine.

Pellagra is a non-contagious endemic disease occurring in adults in parts of Italy, Spain, and the Tyrol, due to eating diseased maize, and characterised by erythema, vesicular and bullous eruptions, pigmentation, atrophy, and progressive constitutional weakness, terminating fatally in 5 to 15 years.

Macular Leprosy appears as brownish or mahogany red patches of erythema of various sizes (§ 490).

c. *Eruptions which usually consist of Papular Elements.*

Common.

I. Acne vulgaris and other forms of acne.
II. Prurigo.
III. Scabies.
IV. Papular syphilide.
V. Skin diseases, sometimes papular at *one stage* :—
    (i.) Papular eczema.
    (ii.) Psoriasis and other scaly eruptions.
    (iii.) Exanthemata.
    (iv.) Pustular and vesicular diseases.
    (v.) Erythematous eruptions.
    (vi.) Nodular eruptions.

Rarer.

VI. Lichen planus.
VII. Keratosis pilaris and follicularis.
VIII. Milium.
IX. Lichen scrofulosorum.
X. Adenoma sebaceum.

§ 462. I. **Acne Vulgaris** is an eruption consisting of "blackheads" or comedones and indurated papules which may go on to suppuration, confined to the face, shoulders, and back, and sometimes the chest. Acne papules are pale red to crimson, and hard (A. indurata), varying in size from a pin's head to a small pea. These are invariably accompanied by a number of black points (comedones), which are black plugs of sebum. The papules come out one after another, and are remarkably persistent; some go on to pustulation, or the formation of small abscesses, leaving scars if untreated. The skin of the face in individuals affected with acne is usually greasy, coarse, and dusky-looking.

Causes. The disease in a considerable proportion of cases starts soon after puberty, and fresh papules may continue to appear for some years, occasionally to middle life or old age. Acne is frequently associated with seborrhœa of the scalp, and if it were not that seborrhœa produces in some cases a characteristic eruption of its own upon the face (see below), seborrhœa of the scalp might be regarded as the source of acne. Inspissated sebum blocking the follicles is the histological cause of the papules, and the change which the hair and sebaceous follicles undergo at puberty appears to be one of the factors in the causation of disease. Several observers have found a bacillus in the papules, which they consider the causative factor. Indigestion undoubtedly aggravates the condition.

Varieties. 1. *Acne punctata, A. indurata,* and *A. pustulosa* are stages in the diseased process, not varieties in the true sense of the word. 2. Associated with seborrhœa of the scalp a crop of papules often appears on the face, almost identical with acne spots, excepting that they are smaller, softer, and not notably accompanied by comedones. This condition may be conveniently distinguished from acne vulgaris by the name "*seborrhœiform acne.*" Its favourite positions seem to be the chin, the furrows below the angles of the mouth, and sometimes between the scapulæ. 3. *Bromide* and *iodide acne* are indistinguishable from each other, and the individual spots resemble acne vulgaris very closely. Comedones, however, are absent, and there is a greater frequency of distribution over the chest and back, though the face is always first affected. 4. *Acne Rosacea* is mainly an erythema, at any rate in its earlier stages (§ 458).

5. *Acne varioliformis* is a severe variety of a. pustulosa. It occurs chiefly on the brow, and is apt to leave deep scars. 6. *A. cachecticorum* (Hebra), or a. scrofulosorum (Colcott Fox[1]), or a. necrogenica is another pustular variety affecting chiefly the back and extensor

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[1] *Lancet*, 1895, vol. ii., p. 499.

surfaces of the limbs, and to a less degree the face, in debilitated or scrofulous children. It is now regarded as a tuberculide. It differs markedly from a. vulgaris in its distribution, and though it may last for many years, is generally amenable to tonic treatment and cod-liver oil. It is liable to be mistaken for a syphilide, but the latter. is less indolent, and there is a firmer infiltration.

The *diagnosis* of acne from the other eruptions in this group is not usually difficult on account of (1) its characteristic position, and (2) the presence of comedones. Papular, pustular, and tubercular syphilides affecting the face are usually copper-coloured, and grouped in a serpiginous manner. Lupus vulgaris generally occupies one side of the face and presents no comedones. Seborrhœa may, as we have seen, give rise to an eruption on the face of discrete papules.

Treatment. If there be much irritation soothing applications are best applied at first, *e.g.*, calamine lotion. Of all remedial agents sulphur is the most efficacious. A sulphur ointment (20 to 40 grs. to the oz.) should be rubbed on night and morning (or F. 95), or a sulphur lotion, or thio-resorcin in strengths varying according to the skin of the individual. For pustular acne mercury is indicated, as in all suppurating affections, *e.g.*, an ointment of 10 to 30 grs. to the oz. To prevent scarring an antiseptic lotion should be used, each pustule must be lanced and its interior wiped out with a pointed stick or match dipped in pure carbolic or camphor phenique. The scalp should always be examined for seborrhœa, and if necessary treated, for unless this be cured the acne may recur. Indigestion aggravates acne, and if present must be corrected. Washing the face frequently with warm water and soap, rinsing off the soap, and rubbing with a rough towel is a valuable domestic remedy. X-rays have recently found favour in some irritable cases.

§ 463. II. **Prurigo** is a disease in which the leading and sometimes the only symptom is generalised itching (pruritus), but it is frequently accompanied by an eruption of papules, urticarial patches, and scratch-marks. The papules of *idiopathic prurigo* are hard, shotty, acuminate, pale red, frequently better felt than seen (giving the sensation of a nutmeg-grater), come out in crops chiefly on the extensor surfaces of the thighs and arms, the trunk, especially the back and buttocks, and only occasionally the face. Each crop lasts a week or two, and is sometimes accompanied by urticarial blotches; dermatographia can generally be elicited. The intense itching leads to scratch-marks. In the course of time prurigo is followed by a dry, rough, thickened, pigmented skin. The *prognosis* is unfavourable, especially in the very poor, the disease recurring for years, and sometimes lasting for life.

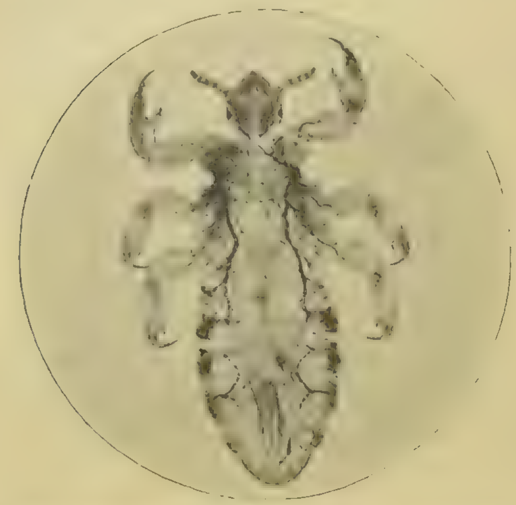

Fig. 132. Pediculus corporis, magnified about 10 times.

The *diagnosis* is simple in well-marked cases by the intensity of the itching and the condition of the skin. The eruption of prurigo very closely resembles that of scabies (§ 464) and pediculi corporis (fig. 132), but in scabies it is almost confined to the *flexures of the joints* instead of the extensor surfaces; in pediculosis to the *shoulders*, where it is attended by typical staining. The pruriginous eruption of pediculi pubis (Fig. 133) is more or less localised. The diagnosis from papular eczema is not always easy; eczema, however, generally prefers the flexures and flexor surfaces, while prurigo predominates on if it is not confined to the extensor aspects.

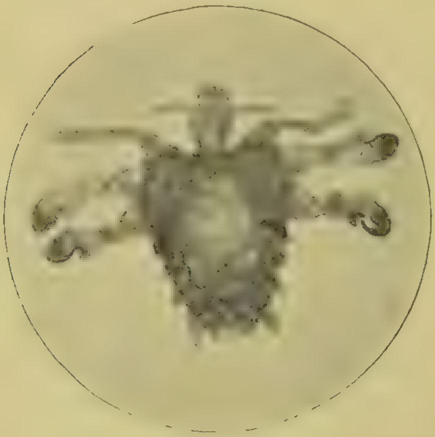

Fig. 133.—Pediculus pubis, magnified about 10 times.

Varieties. (1) **Prurigo infantalis** (syn. urticaria papulosa, lichen infantalis, lichen urticatus, prurigo mitis) should, in the author's opinion, be classified as a variety of prurigo. The papules are small, chiefly on the back, and the urticarial element moderate. It starts about the 4th month of life and recurs until about the 4th year. (2) In **P. adolescentium** the papules are larger, the urticaria prominent, the skin brown and thickened. (3) In **P. senilis** (syn. pruritus senilis) the eruption may be insignificant or absent, and the irritation intractable, with a tendency to induration and purpuric complications. (4) In "*pruriginous*" *eczema*, an eczema complicates a prurigo; (5) in *P. agrius*, all the lesions are on a larger scale, the inguinal glands involved, and the general health seriously damaged. (6) *Summer prurigo* (syn. lichen æstivalis) develops in hot weather, and the papules tend to vesiculation (hydroa vacciniforme); and (7) *P. hiemalis* occurs in cold countries or in the winter only.

Etiology. Prurigo is especially apt to occur at the two extremes of life, and is probably a blood disorder with irritability of the cutaneous nerve endings.[1] Irritation of the alimentary tract is liable to start outbursts of the disease, which is greatly influenced by improper food, and unhealthy surroundings. It may be idiopathic, or secondary to scabies or pediculi. The idiopathic causes of prurigo are the same as those of pruritus (§ 449c).

Treatment. The first indication is to discover the cause, for without carefully sifting this question no treatment can be successfully carried out. One must first exclude phtheiriasis, scabies, or other parasites. Then determine whether there is any other source of local irritation, such as an unbleached cotton or flannel garment, or an acrid discharge; and finally turn to the idiopathic causes. Warm weak tar baths should be taken night and morning, followed by the inunction of a mild tar or mercury ointment, to get the skin into a healthier condition, and the softest of undergarments should be worn. For local itching carbolic acid 1 in 40, alkaline lotions, vinegar, chloral-camphor (ʒj. of each liquefied and added to ℥j. starch powder) are all useful. The diet should be plain; sugar, alcohol, and excess of meat must be avoided. Any digestive errors should be corrected, and the possibility of mucous (catarrhal) colitis borne in mind. Calcium chloride in large doses has been very efficacious in my experience (*loc. cit.*). Other remedies which are worth a trial are—cannabis indica, antipyrin, valerian, bromides (especially in nervous subjects), pilocarpin, liq. am. acet., atropine, carbolic acid, salol, and other intestinal antiseptics.

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[1] "The Pathology of Itching, and its Treatment by Large Doses of Calcium Chloride," *The Lancet*, Aug. 1, 1896, p. 300.

§ **464.** III. **Scabies** is the eruption produced by the acarus scabiei. It consists of papules and vesicles of varying size; occasionally the latter go on to the formation of pustules. In addition to its multiform character, it is readily diagnosed in its typical form—(1) by the short white or black burrows; (2) by the excessive itching, and scratch-marks; (3) by the fact that it always commences and predominates where the skin is thinnest, *i.e.*, between the fingers or toes, the flexures of the wrists and elbows, the axillæ, the penis, the inner side of the feet and thighs; (4) by history of contagion; and (5) lastly, by the discovery of the animal or its eggs (Fig. 134). Prurigo is likely to be mistaken for scabies, but prurigo predominates on the extensor surface and outer side of the limbs. Eczema is perhaps the disease with which, in the adult, it is most frequently confused. Many experienced physicians have overlooked the parasitic origin of eczema thus produced, and it is a good rule to suspect scabies in inveterate cases of eczema. Scabies untreated may go on for an indefinite time, but it readily yields to treatment. Sulphur ointment should be thoroughly applied after a warm bath with plenty of soap, for at least 4 successive nights, and the underlinen and sheets should be boiled. Balsam of Peru, either pure or mixed with equal parts of vaseline, is a more expensive but equally efficacious remedy. If the sulphur produce a secondary eruption, mix it with equal parts of zinc ointment.

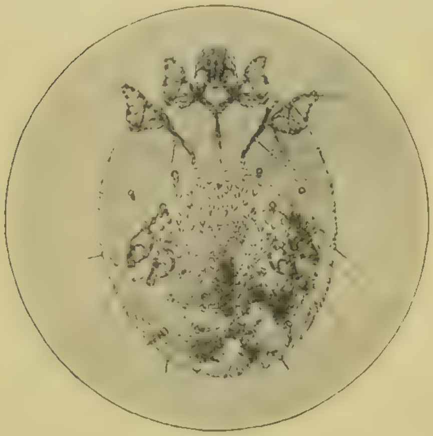

Fig. 134.—ACARUS SCABIEI (female), magnified about 20 times. The female burrows under the skin to lay her eggs, the burrows thus formed being quite typical and black with the fæces of the insect; most frequently situated on the wrist. The male roams about over the body and clothes and is rarely captured.

§ **465.** IV. **Papular Syphilide.** Syphilitic eruptions are often multiform, but papules generally form the most prominent feature of all syphilitic rashes, especially in the secondary stage. The papule, indeed, forms the prototype of all syphilitic eruptions. These papules are firm, glistening, and project above the surface of the skin with a hard, infiltrated margin, and *vary in size* from a pin's head to a bean. They are of a brownish-red colour (like copper or raw ham) which does not entirely disappear on pressure. The wide variability in the size of the papules is a feature distinguishing this from other papular diseases. As they increase in size the centre often becomes depressed, or cupped. The distribution is more or less generalised, and often symmetrical, but the favourite seats are the forehead, around the mouth, the *flexor aspects* of the arms, and the trunk. When near the corners of the mouth or the anus their surface may be moist, and the *secretion is highly infectious*, no matter how long it is since the primary malady was contracted. Itching is usually absent. Other constitutional signs of syphilis may be present. The presence of shotty glands in the groin and neck and elsewhere is a valuable aid in the diagnosis of all syphilitic eruptions. They are present even when no other signs are present, and may last throughout the patient's life. Two *varieties* of papular syphilide are described according to the prevailing size of the elements, *papular syphilide* if the spots are small and numerous, *lenticular syphilide* if they are large and scanty. The former is met with more in the early, the latter in the later stages of the disease. A rarer form is the *corymbose syphilide* in which there are clusters of small red rounded papules scattered over the body and limbs, which leave corresponding groups of atrophic scars. See also §§ 405 and 408.

V. **Skin Diseases sometimes papular.** ECZEMA PAPULOSUM (papular eczema, pruriginous eczema) is a term which should be confined to papules which rapidly pass on to vesiculation, or which are associated with definite patches of eczema. Papules frequently form a stage, generally an early stage, in PSORIASIS, SEBORRHŒA, PITYRIASIS RUBRA PILARIS, SYCOSIS, in the EXANTHEMATA, ERYTHEMATA, etc., and will be dealt with in their proper place.

Rarer Papular Diseases.

§ 466. VI. **Lichen Planus** (synon. Lichen Ruber Planus) is an eruption consisting of flattened, angular, shiny, dull red papules, often presenting a central depression, and a small silvery scale on the surface. These tend to coalesce and form irregular patches of a peculiar purplish hue. Occasionally rings are formed (lichen annularis). There is never any exudation. When the papules disappear, a good deal of pigmentation may be left behind. The eruption is very frequently symmetrical, and by far the most characteristic positions are the flexor aspect of the wrists and forearms, and the inner side of the knee. Sometimes the distribution is more generalised; and the mucous membrane of the mouth may be affected. Itching as a rule is present, and in some cases is very troublesome. By the fusion of several papules large plaques may be formed, and when these take on a warty growth, as about the ankles, the condition, which is very intractable, is called *lichen verrucosus*. Lichen occurs mostly about middle age, and has been noticed to appear when there is a degree of nervous debility. In Vienna two-thirds of the patients attacked are men (Kaposi), æt. 10 to 40; whereas in England the majority of cases occur in women. There should be no difficulty in *diagnosing* lichen planus from a *papular syphilide* or an *eczema*, on account of its typical positions, angular shape, purple colour and flat waxy top of the papules. The *prognosis* as to the cure of the condition is good under treatment, although this may have to be extended over many months. The eruption remains localised to definite regions for years, and rarely becomes generalised like lichen ruber acuminatus. The *treatment* consists in the administration of arsenic internally, and local soothing applications, such as F. 90 or 93 (see also § 498). Some recommend small doses of hydrarg. perchlor.

§ **467.** VII. **Keratosis Pilaris** (syns. Pityriasis pilaris; Lichen pilaris; Lichen spinosum) is an affection of the skin, generally of young and vigorous adults, in which the orifices of the hair follicles of the thicker portions of the skin, *i.e.*, on the extensor and outer surfaces of the limbs, are occluded with corneous plugs. These give to the skin the appearance of being covered with tiny sharp hard spines. The pathology is obscure, but hard friction with almost any ointment at night and a rough towel in the the morning will generally remedy the condition in the course of a few weeks.

Keratosis Follicularis (syn. Darier's disease; psorospermosis) is a very rare disease, due to overgrowth and degeneration of cells in the mouths of the pilo-sebaceous follicles. The papules are at first of pin-head size, resembling keratosis pilaris. They contain in the centre a horny plug, which is difficult to remove. Some become enlarged and hyperæmic; others become confluent, presenting a papillomatous surface covered by hard yellowish crusts. These may ulcerate, and the area may be covered with a mucopurulent discharge. The disease affects first the face and head, and after the gradual development of years, appears over the sternum, spine, loins, hypogastric and inguinal regions and the extremities, with symmetrical disposition. The *diagnosis* may be difficult, in the early stages, from lichen planus and ichthyosis; but in these diseases there is no central horny plug; and in the later stages from acanthosis nigricans. *Treatment* consists in the use of salicylic, sulphur, or other keratolytic applications.

§ **468.** Three rare conditions—Milium, Lichen scrofulosorum and Adenoma sebaceum—come under the papular rashes.

VIII. **Milium.** The term milium is applied to an eruption of small whitish or yellowish pearly granules about the size of a pin-point which affects chiefly the delicate skin under the eyes, the eyelids, cheeks, temples, scrotum, and labia. It is due to retention within the sebaceous follicles owing to an overgrowth of the stratum corneum closing their openings. Some say they consist of embryonic tissue. The *treatment* consists in making a small incision over the granule, and squeezing out the contents. Or electrolysis may be employed.

IX. **Lichen Scrofulosorum** (synon. Lichen ruber scrofulosorum; L. circumscriptus; L. piliare des strumeux (Besnier); Perifolliculitis tuberculosa; Tuberculosis cutis lichenoïdes) is an eruption of minute yellowish-red papules, isolated or grouped, and occurring usually on the breast, abdomen, or back of tuberculous subjects. They may be slightly scaly, are painless, and do not itch. It is not common after the age of 20. It is not serious, and may last, hardly noticed by the patient, for months or years, and then subside without leaving a trace behind it. Its treatment is that of other tuberculous affections. It is probably the same disease as acne necrotica.

X. **Adenoma Sebaceum** is a rare disease, consisting of numerous small hemispherical elevations, discrete, grouped usually about the middle of the face. In size they vary from a pin-head to a split pea. Their surface may be smooth or rough; they are crimson or pinkish yellow, and associated frequently with telangiectasis. They have no visible orifice, but if incised, portions of sebaceous gland and matter can be expressed. Some disappear spontaneously, leaving a small scar. The disease is almost always congenital, though it may not be observed till puberty, when it takes on fresh activity. It is said to be associated with intellectual inferiority, but this is not always the case. It is due to overgrowth of the sebaceous glands and hair follicles. The knife, electro-cautery, or electrolysis are necessary to destroy the growths where spontaneous involution does not occur.

d. Eruptions usually scaly or scurfy.

Common.

I. Psoriasis.
II. Seborrhœic dermatitis.
III. Scaly syphilide.
IV. Skin diseases sometimes scaly at one stage, *e.g.*, eczema, lichen, erythematous diseases.

Rarer.

V. Exfoliative dermatitis.
VI. Pityriasis Rosea.
VII. Pityriasis rubra pilaris.
VIII. Ichthyosis.
IX. Erythrasma.

§ 469. I. **Psoriasis** is a common disease, occurring as irregular ovoid patches, slightly raised, covered with copious silvery scales, unattended by any exudation, and situated chiefly on the elbows and knees. The lesion starts as a tiny papule which from the first has a scale on the top (P. punctata); this gradually becomes larger, and is called P. guttata. In a short time it reaches the size of a coin (P. nummularis). The disease generally then remains stationary for some weeks or months, and may tend to undergo spontaneous involution. The healing process usually starts at or near the centre and gives to the eruption a circular or serpiginous appearance (P. circinata, P. gyrata). The lesion is scaly and elevated from the first and always dry, three features which at once distinguish it from eczema, and if the tops are scratched off bleeding hyperæmic papillæ are exposed. The distribution is extremely characteristic, being found always on the knees and elbows, frequently on the scalp, trunk, and other parts of the limbs, and only very rarely on the face, palms, or soles. There is little or no itching or subjective symptoms.

Causes. As regards age, the disease almost always occurs in early life, though rarely before seven years old. Both sexes are equally affected. There is a considerable hereditary predisposition in some families. The seasonal influence varies in my experience, but those who are subject to the malady often complain that it recurs each winter or spring.

Diagnosis. It is important to distinguish psoriasis from *scaly syphilide*, and in many cases the character of the lesion affords no true guide. A scaly syphilide is not confined to the elbows and knees and generally prefers the flexor aspects, and palms and soles; the centre of the patches are usually depressed, stained, and healing the scales are scantier, less silvery, and on being scraped off do not leave bleeding points. *Seborrhœic dermatitis* is sometimes difficult to differentiate from psoriasis, and indeed Unna (the describer of this disease) holds that they are identical. The seborrhœic patches are less crimson, the scales smaller, scantier, greasier, and more orange coloured; they occur by preference on the shoulders and upper parts of the trunk, and if affecting the limbs are usually on the flexor aspects. The scalp may be affected in both diseases.

Prognosis. Psoriasis is apt to disappear and to recur spontaneously at certain seasons. The patient may take little heed of the disease unless it affect the uncovered parts. In severe cases the eruption may spread over the whole body.

Treatment. Psoriasis is essentially a chronic malady, and the indications are to remove the diseased parts and stimulate a healthy growth. Chrysarobin ($\frac{1}{2}$ to 1 dr. to the oz.) is the most valuable remedy. The chief objections to its use are that it stains the linen and the skin and frequently sets up a scarlatiniform dermatitis. Other stimulating applications, such as tar, carbolic acid, and salicylic acid are often quite as efficacious. Plaster mulls of the same ingredients, or of mercury, are useful. For intractable cases I am in the habit of using chrysarobin paint—20 per cent. in chloroform—painted on, and covered with a layer of collodion. This is applied once a week. Internal medication often does good. Thyroid extract has not fulfilled the expectations of its advocates. Arsenic, cod-liver oil, salicylates (Crocker), and copaiba (M'Call Anderson) are recommended.

§ **470.** II. **Seborrhœic Dermatitis** (syn. Eczema Seborrhœicum (Unna)) was first described by Unna. It occurs in irregular ovoid patches, greasy, covered with brownish-red scales; quite superficial, sometimes slightly raised, with sloping margins. They sometimes heal in the centre, forming a ringed eruption which may be mistaken for ringworm. The patches vary in size from a small pea to a crown piece, or larger. Single papules may be present, brick-coloured, and soft, especially on the face. The disease is found *chiefly on the scalp* whence it may spread on to the forehead and face, then on the sternum and back of the neck and shoulders. It is often limited to the upper part of the body, but the lower parts of the body may be affected, and the legs occasionally present typical patches. The affection is more frequent than is generally thought, being often diagnosed as ordinary eczema when from irritation or other causes it passes on to an eczema with vesicles and crusts. It is frequently associated with psoriasis ; Unna and others consider these two diseases identical (*vide supra*). The author does not share this view. The disease is more frequent in, though not confined to, young people. It is undoubtedly contagious in the author's experience, and is particularly apt to spread to those who occupy the same bed and whose pillows are apt to be exchanged. Professor Unna ascribed it originally to the bottle bacillus, Dr. Sabouraud to the staphylococcus griseus.

The *treatment* must not only be directed to the cure of the patches which are on the body, but to the scalp, for until this is cured the eruption will constantly recur upon the body. A pomade of hyd. ox. rub. (gr. 7 to 10 ad ℥j.) should be rubbed in twice a week, or a lotion of hyd. perchlor. (5 grs. to the oz.). Sulphur is equally efficacious, but is a disagreeable application. See also § 497.

Epidemic Peri-oral Eczema. In 1895 the author had the opportunity of investigating an eruption which occurred on the faces of a large number of children in one school, in the east end of London. The school contained nearly a thousand children and quite half of these were affected in succession during three or four months. The patches occurred mostly around the mouth, on the face and neck, and rarely elsewhere. They were superficial, reddish, dry, scurfy, ovoid, somewhat resembling a superficial seborrhœic dermatitis. They were supposed to be ringworm, but this was positively excluded. The disease readily yielded to a mild tar and mercury ointment. It certainly spread from child to child, but only one of the teachers was slightly affected. (*The Lancet*, vol. i., 1896.)

§ **471.** III. **Squamous Syphilide** (syphilitic psoriasis). The squamous syphilide occurs in the later stage of the papular or the tubercular syphilitic eruptions (*q.v.*), and does not constitute a separate form of eruption of itself. It is recognised by the fact that the scales are thin, scanty, and greyish, lying upon patches of stained or infiltrated skin (*i.e.*, the syphilitic papule) which are deep brown or copper coloured, usually round, or in the shape of *segments of circles*, having raised serpiginous scaly borders. A squamous syphilide may occur on any part of the body, but the flexor aspects and the palms or soles are particularly characteristic situations, the converse of psoriasis vulgaris. A scaly syphilide of the palms is diagnosed from dry eczema by its raised serpiginous border, with sometimes an area of healthy but pigmented skin in its centre.

IV. Certain skin diseases are scaly at one stage. A *scaly* or scurfy condition of the skin, especially of the face, is produced by hard water and exposure and in certain states of ill health. It is also met with after scarlatina, measles, and some of the other eruptive fevers. In *eczema*, which is a somewhat protean disease, scales and crusts form, but the presence of exudation is its essential and differentiating quality. *Pityriasis capitis* is a scaly or scurfy condition of the scalp, sometimes called also seborrhœa sicca, or dandruff (see diseases of the scalp). In several varieties of *lichen*, a thin silvery scale is constantly found, although they belong really to the papular eruptions. This occurs also in papular syphilide, and it is difficult sometimes to draw the line between a papular and a scaly syphilide. *Lupus erythematosus* is attended by adherent scales and crusts.

§ 472. V. **Exfoliative Dermatitis**. Much discussion ranges around the use of this term and the term Pityriasis Rubra, which has been employed by some as a synonym. It is best to regard the term Exfoliative Dermatitis as implying any chronic or sub-acute generalised inflammatory disease of the skin, whether primary or supervening upon other cutaneous disturbance of long standing, which is characterised by hyperæmia of the entire surface and *abundant and repeated exfoliation* of the cuticle, accompanied usually by shedding of the hair and nails. There is usually some constitutional disturbance, and the itching may be severe.

Etiology. Occasionally, as a *secondary* affection, it may follow psoriasis, eczema, pityriasis rubra pilaris, pemphigus foliaceus and seborrhœic dermatitis. As a *primary* condition the disease is of considerable gravity. It starts in several different ways, though a rapidly spreading hyperæmia of the integument is common to all. It is my own belief that the different varieties which have been described as separate diseases only differ really in their mode of onset and etiology. In the *treatment* of the malady general tonic and other internal treatment, as a rule with stimulants, occupies a prominent position. This should be combined with creolin and other soothing baths and ointments.

An **epidemic exfoliative dermatitis** which the author first had the opportunity of observing in 1891[1] illustrated very well the wide varieties both in the severity and other features of this malady. 163 cases occurred among the patients in the Paddington Infirmary with a case mortality of 12·5 per cent. That the disease was epidemic and contagious was certain, that it was microbic seemed probable; though the author's later observations on epidemics at other workhouses and infirmaries tend to show that the milk consumed by the patients was in some way the means of propagating the disease amongst them, possibly having undergone some toxic or fermentative change.

§ 473. VI. **Pityriasis Rosea** consists of numerous pink patches, slightly raised, varying in size from a pea to a florin. There is no infiltration; slight scaling is often present at the margin of the patches. They spread peripherally, and many present a circinate appearance. The eruption starts usually on the sides of the trunk, and may spread to the neck, upper arms and thighs. It is rare on the face. Slight itching may be present. Pityriasis Rosea runs a course of a few weeks to a few months, and disappears spontaneously. *Etiology.* The disease occurs in both sexes, and at any age, but is most frequent in young adults. It is believed to be of parasitic origin, and some hold that it is feebly contagious. *Seborrhœic eczema* has a more gradual course, and has greasy scales. *Tinea circinata* occurs in fewer patches, and the fungus can be found. *Psoriasis* has more infiltration and diffuse scaling. *Syphilis* has a darker colour and is infiltrated.

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[1] "Monograph on Epidemic Skin Disease," H. K. Lewis, London, 1892; Med. Soc. Trans., 1891; *Brit. Med. Journ.*, Dec., 1891.

Treatment. Mild ointments of sulphur or salicylic hasten the course of the disease, and soothing lotions are useful if itching is present.

§ 474. VII. **Pityriasis Rubra Pilaris** (Devergie), Lichen Acuminatus or Lichen Ruber (Hebra), is a somewhat rare disease in which the eruption commences as tiny papules involving the hair follicles, which become fused together into one reddened surface, and shed a succession of flaky scales. The distribution is fairly characteristic, as it starts where the lanugo hairs are mostly found, namely, the backs of the hands and forearms. In this way it often presents a glove-like distribution on the upper and lower extremities which is very characteristic. It may spread over the whole body. The progressive margin is always marked by the same tiny scale-capped papules. The disease has to be *diagnosed* from psoriasis on the one hand and dermatitis exfoliativa on the other. It is differentiated from *psoriasis* by its distribution and by the presence of the little papules at the spreading margin of the eruption, but is indistinguishable from dermatitis exfoliativa when the whole body is involved, except by the large flakes of epidermis in the latter. In the earlier stages, dermatitis exfoliativa does not present the small acuminate papules which constitute the elementary lesion of P. rubra pilaris.

The *causes* are obscure. The disease usually occurs before the age of 21. *Prognosis.* The malady may occur in varying degrees of acuteness. Some cases are ushered in with a certain degree of constitutional disturbance, vomiting, and some pyrexia, and after lasting a few months will tend to spontaneously subside. In mild cases there are no constitutional symptoms, and the disease runs a prolonged course of many months. *Treatment.* When it occurs in the chronic form arsenic is a remedy of some value. The same rules guide us as in the treatment of eczema.

§ 475. VIII. **Ichthyosis** (syn. Xeroderma) may be defined as a congenital condition of the skin, characterised by an undue dryness, roughness and scaliness of the epidermis, and in some cases by the formation of wart-like outgrowths. Though congenital, the condition may not be identified till the child is some years old.

There are three *clinical types* or degrees of the affection. In the first or mild type (*Xeroderma*) there is simply an undue harshness or roughness of the skin, and consequently through life a great tendency to the supervention of "chaps," eczema and other skin affections. It occurs chiefly on the extensor aspects. In a second type (*I. vera*) the superficial layers of the epidermis are thickened and present the appearance of being stretched. The hardened cuticle which results presents fissures and cracks which, bounding polygonal areas, give to the patient the appearance of a fish or crocodile skin. The everted eyelids and nostrils, the atrophied hair and nails and the hardened, scale-like condition of the skin give a very characteristic appearance. In the third variety (*I. hystrix or I. sebacea*) the skin in certain places presents a thickset aggregation of little horny, wart-like processes, which entangle the dirt and present a brownish-black coloration (Fig., p. 844). These are arranged in streaks, which were believed to follow the course of certain nerves, but a closer observation shows that this is not so—the disease being a developmental one. The diagnosis is not difficult, owing to the congenital nature of the malady. Apart from the inconvenience, and the great liability to eczema and other morbid conditions of the skin, the first-named type is not serious. In the second type the disease progresses to the age of puberty, if the child lives so long, and then remains stationary. The third type rarely shortens life, but is a disfiguring malady. No known remedy influences this disease. Vaseline, lanolin, baths and various ointments may soften the skin and remove the superficial scales to some extent. Thyroid seems to control milder cases to some extent.

§ 476. IX. **Erythrasma** consists of defined scaly discs, of a pale red, yellow or dark brown colour, occurring on the skin of certain hairy parts. The scales can be scraped off,

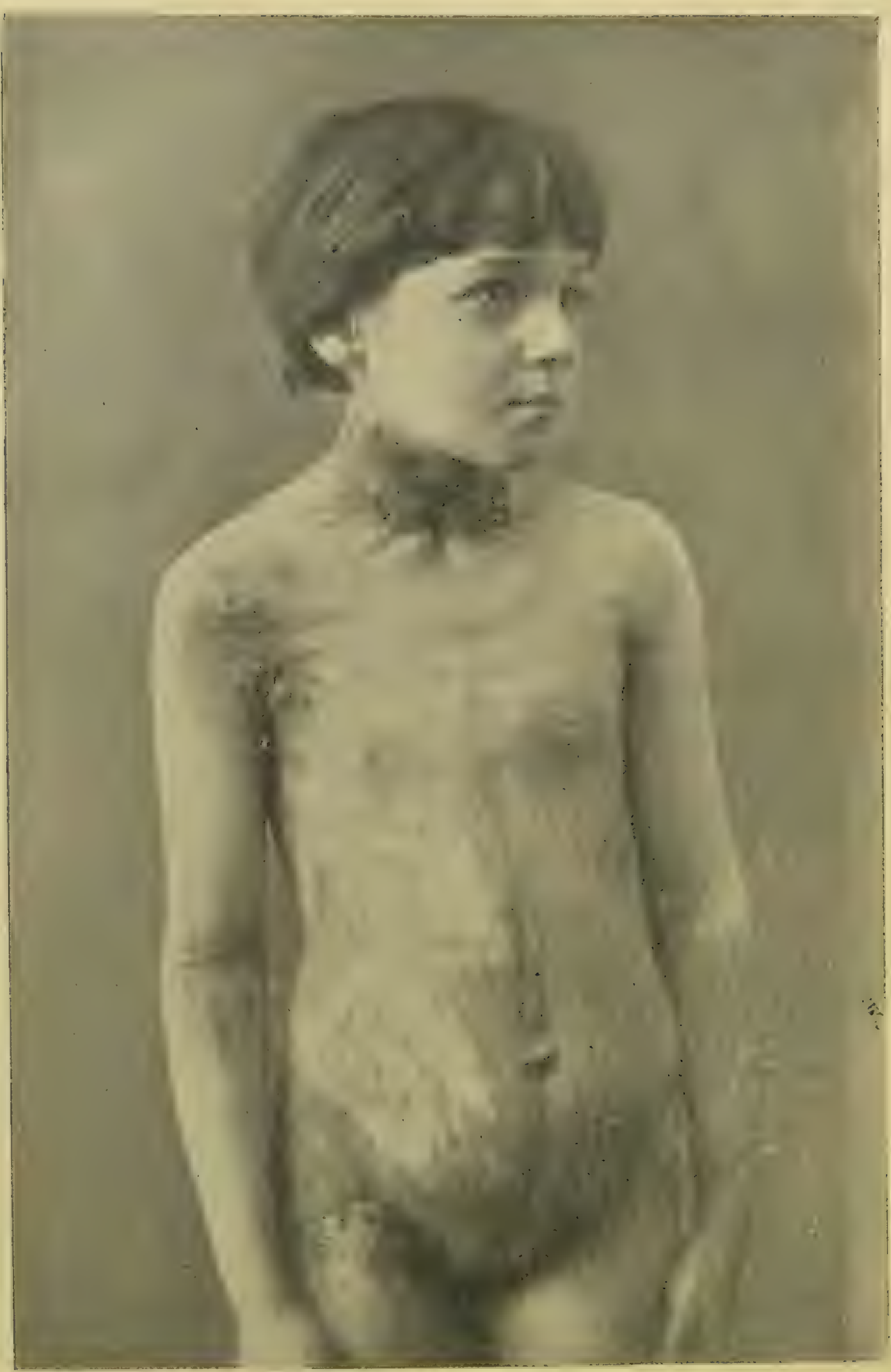

Fig. 135.—Ichthyosis Sebacea in a girl 14 years of age. The linear distribution of the dirty, wart-like excrescences is quite characteristic. This condition is probably identical with (or allied to) I. hystrix and Neuro-papilloma. See p. 843.

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and are found to contain a fungus, the microsporon minutissimum. The patches are extremely chronic, and are found on the opposed surfaces of the scrotum, thighs, axillæ, and mammæ. They itch only when perspiration is excessive.

GROUP II. VESICULAR AND WEEPING ERUPTIONS.

Moist eruptions, in which the elements are usually vesicular and the exudation serous, are commonly classed into those with small vesicles (I. to VIII. below), and those with vesicles of larger size, bullæ (IX. and X. below).

I. Eczema.
II. Impetigo contagiosa.
III. Herpes.
IV. Varicella.
V. Scabies.
VI. Tinea circinata (sometimes).
VII. Sudamina.
VIII. Hydrocystoma.

IX. Dermatitis herpetiformis.
X. Pemphigus.

XI. Pustular and other diseases in which vesicles and bullæ may occur at some stage.

NOTE.—Syphilides are practically never vesicular.

§ 477. I. **Eczema** is a catarrhal inflammation of the skin, running sometimes an acute, sometimes a chronic course, presenting a red excoriated surface denuded of its epithelium and more or less covered with crusts, associated in its acute stages with non-marginated swelling. "Weeping," *i.e.*, a serous exudation which stiffens linen, is the chief characteristic of eczema. Although eczema has been defined as a vesicular disease, it may present different appearances at different stages of its course. In eczema are seen, at different stages, the three primary and three secondary lesions of the skin—erythema, papules, vesicles, crusts, scales, and fissures. In the first stage, or *acute* eczema, there is erythema, with papules and tiny vesicles, which readily rupture, and the serous exudation results. By the time the physician sees the case, *e.g.*, in a day or two, the second or *subacute* state is usually reached, with excoriations and crusts, and the involved patch of skin presents a more or less swollen surface, denuded of its epithelium. If the disease passes on to the third or *chronic* stage, the discharge decreases or disappears, leaving a thickened, irregular, scaly patch, fading at its margins. Any part of the body may be affected, and to any extent; but eczema has a predilection for the flexor aspects of the limbs and the flexures of the joints. The patient complains of a burning, smarting, throbbing, or itching, in proportion to the acuteness of the process.

The *diagnosis* of eczema is not difficult. *Seborrhoic dermatitis* has no serous exudation and is covered by greasy yellow scurf. *Syphilides* never resemble acute or subacute eczema, or indeed any vesicular disease, a fact of considerable value in practical diagnosis. It is difficult sometimes to distinguish patches of dry chronic eczema from *psoriasis*, but the latter affects characteristic localities, preferably the extensor aspects, and is covered with silvery white scales. The diagnosis of the numerous varieties will be given below.

The history or evidence of a *cause* should be sought. (1) *Local causes.* Eczema is the lesion most frequently following the dermatitis produced by local irritants, such as a mustard plaster, turpentine, the leaves of certain plants (*e.g.*, the primula obconica), or soaking the hands in water containing soda. Eczema forms around the eyes treated with eye-drops; and excessive sweating may produce the condition. It often occurs around the mucous orifices, from which an irritating discharge issues (the nose, ears, anus, etc.), and under these circumstances is very intractable. The local hypostatic congestion attending varicose veins is a frequent predisposing factor. Unrecognised scabies or pediculi lead to protracted cases of eczema of multiform character. (2) *Constitutional causes.* Eczema may occur as a complication of dyspepsia, gout, diabetes, or renal disease. In cases of eczema of the vulva or prepuce, the urine should be examined for sugar, and the presence or absence of leucorrhœa ascertained. It often accompanies albuminuria, especially if dropsy be present. It may appear during every pregnancy, or after the cessation of lactation, when neuropathic influences are at work.

Varieties. In addition to the typical acute and chronic forms described, there are several varieties of eczema. (1) In *E. papulosum* the process stops at a papular stage. (2) *E. pustulosum* or *impetiginodes* is a pustular form resembling impetigo, due to pyogenic cocci, occurring frequently on the heads or faces of children, on the hairy parts of delicate and tuberculous persons, or when any local irritation is present: it is attended by the formation of crusts. (3) *E. rubrum* occurs usually on the legs, chiefly in old people, where the deficient circulation produces a livid colour, and prevents repair. It may resemble pemphigus foliaceus. (4) In *Vesicular E.* the vesicles are prominent, tend to come in crops, and become confluent. Its favourite localities seem to be the face, the ears, and the flexures of the limbs, fingers and toes. Some cases of this disease resemble dermatitis herpetiformis. (5) *E. squamosum* is a chronic scaly stage into which many erythematous and papular varieties develop, and is found most frequently on the palms, legs, and scalp, and is apt to be mistaken for psoriasis and squamous syphilide. (6) Occurring in different parts of the body, eczema is often named from the locality—E. capitis, E. ani, E. intertrigo, but it is unnecessary to invent special terms for these varieties, except perhaps in the case of *E. palmaris*, which is often due to the sufferer's occupation (baker's itch, washerwoman's itch, etc.). (7) In the palms it is apt to become chronic, dry, thickened, and fissured (*E. rimosum*).

(8) *Cheiro-pompholyx* is the term given to a vesicular and bullous eruption affecting the hands *symmetrically*, and sometimes the feet at the same time. The thickness of the epidermis in this situation prevents the rupture of the vesicles. These are especially prone to appear in the clefts between the fingers and toes, like boiled sago grains, and creep on to the palmar and dorsal surfaces—an important diagnostic feature between this disease in its later stages and a scaly syphilide of the palms. Some of the vesicles coalesce into bullæ, their contents become absorbed, and exfoliation of the epidermis occurs.

(9) *E. Marginatum* is tinea circinata of the groins and genitals.

(10) *Paget's disease* of the nipple is really a malignant form of eczema. It starts on the nipple of one or both mammæ, and spreads centrifugally, sometimes with a slightly raised margin, leaving a reddened, congested, scurfy, and sometimes weeping surface. It is met with mostly in females of advancing life, and it consists of a slowly-growing cancerous process.

(11) *Peri-oral Eczema* is a term which is applied by the writer to an epidemic condition which he observed in a large board school in 1895 (see § 470).

The *treatment* of eczema differs materially according to the stage of the disease. The principles of treatment are those underlying the treatment of all diseases of the skin. In the acute inflammatory stages, when there is much erythema or vesiculation the indication is rest indoors or in bed if necessary, with such soothing applications as zinc oxide, calamine, lead, or bismuth, especially in the form of lotions, powders, and occasionally ointments. Lead or weak creolin lotions are comforting; and for acute eczematous conditions I have found nothing more successful than prescription (F. 42), or lot. calamin. co. Saline aperients are useful; and vin. antimon. ♏x. with mag. sulph. ½ dr. t. d., if used early, seems to sometimes cut short the disease. A daily bath (say a teaspoonful of creolin to 15 gallons of warm water) is indicated whenever the eruption is widespread, and among out-patients in whom want of cleanliness is a potent causal factor. In subacute stages a stimulating agent should be added to the soothing remedy; and in the chronic stages change of air (to hilly country in preference to seaside) and stimulating remedies. These are tar, mercury, resorcin, creosote, carbolic acid, salicylic acid, and sulphur. But a mere knowledge of the drugs to be employed is not sufficient; it is necessary to follow certain rules in their application. Thus for weeping surfaces lotions, not ointments, should be chosen; for thickened surfaces ointments should be not merely smeared on, but spread on a piece of lint, and firmly applied to the skin, so that a macerating effect may be obtained. The same results may be obtained and the part protected by the use of medicated plaster mulls, the most useful of which perhaps is a weak tar and mercury plaster (4 per cent. of the former and ¼ per cent. of the latter). It is of no use to apply remedies over thickened crusts or scabs; these must first be removed by means of bread or starch poultices, or wiped away with olive oil. When the surface is thus cleaned it must be kept aseptic by excluding the air, and renewing the applications every four hours for acute and once or twice a day for chronic conditions. Lotions should rarely be covered with gutta-percha tissue to keep them moist, because the part becomes sodden. Eczematous parts must never be washed with soap and water, but when ointments are used they may first be cleansed with sweet oil or creolin lotion (ʒj. to 1 pint). In the latter stages an ointment containing mercury or tar may be used (*e.g.*, F. 101), the stimulating agent being cautiously increased. If there is much secretion an astringent lotion (such as lead or creolin) may be first employed to cleanse the part, while if the part is dry and scaly, the tar and mercury may be increased without danger. In cases of long duration in which considerable thickening exists, our chief object should be to remove the products of disease so that the healthier underlying structures may resume normal growth. This may be done by the active application of exfoliating remedies such as salicylic acid (20 to 30 grs. or more to the oz.) and sulphur. In very inveterate cases the previous application of strong plaster mulls (*e.g.*, salicylic acid 10 to 30 per cent. and creosote 10 to 40 per cent.) facilitates the process, though it may at first appear to make it worse by setting up an acute inflammation. Tonics and internal remedies should be administered, such as iron, alkalies and gentian, etc., to correct any morbid general condition; arsenic should seldom be used except in chronic non-inflammatory cases. In the intractable eczema affecting the extremities of the aged, I have found small doses (1 to 3♏) of opium of great use. Constitutional treatment is specially indicated when the eruption is widespread or generalised. The digestion must be put in order, and when there is any marked tendency to erythema or congestion three remedies are of great use, alkaline carbonates, quinine and calcium chloride, all in fairly large doses.

Treatment of Varieties. E. of the *eyelids* requires careful treating, lest the ointment should light up conjunctivitis. The following ointment—hyd. ox. flav. and acid borici, āā gr. ij., aq. dest. and ol. amyg. dulc. āā gr. xxx., lanolin 1 oz.—may be found useful. For eczema around *mucous orifices*, the great indication is to keep them dry, and a powder consisting of equal parts of zinc oxide, bismuth carb., and calamine frequently dusted on gives relief. Calamine lotion is also useful. For *E. papulosum* a creolin bath and the removal of the offending garment suffices; for *E. impetiginodes* ung. hyd. am. is almost a specific; *cheiro-pompholyx* is treated on the same lines as acute E.; in *E. palmaris* gloves saturated with the ointment should be worn at night, and if possible by day, and a small quantity of ointment should be smeared on after every washing; *Paget's disease* is an indication for removal of the breast. See also § 498.

II. **Impetigo contagiosa** consists of discrete vesicles of varying size which soon become pustular (§ 483).

Streptococcal and staphylococcal skin infections give rise to many different kinds and sizes of vesicular eruptions of the impetigo class which may be mistaken for eczema or herpes on the one hand, and pemphigus or urticaria bullosa on the other.

§ 478. III. **Herpes** (syn. herpes zoster, zona, shingles) may be defined as an acute non-contagious disorder, consisting of one or more *clusters of vesicles* on a crimson base, associated with neuralgic pain, and due to an irritative lesion of one of the ganglia of the posterior spinal roots or their analogue the Gasserian. Herpes commences with a red patch or a group of flat papules, on which vesicles very rapidly appear. The vesicles are larger than those of eczema, round, hemispherical, and uniform in size, about as big as a millet seed; and as there is no tendency to spontaneous rupture there is usually no oozing such as occurs in eczema. They smart or burn, and the neuralgic pain which precedes and follows the affection is often very severe and intractable. The vesicles contain clear serum, and after lasting a few days dry up and form little crusts. The whole attack lasts on an average 3 weeks. The vesicles leave no ulceration and no scars. The affection is nearly always unilateral. Formerly a patch of herpes was thought to correspond with a sensory nerve distribution, but the groups correspond more precisely with what is known as a sensory area; that is to say, an area which has been shown by Mackenzie, Thorburn and Head to represent the terminal distribution of the pain-appreciative (algetic) fibres connected with each spinal segment (§ 557). Many *varieties* are named according to their position, *e.g.*, H. frontalis, ophthalmicus, brachialis, etc. H. febrilis (facialis or labialis) often accompanies inflammations of the respiratory tract. H. progenitalis, or preputialis, which occurs on the genital organs of both sexes, sometimes alarms patients with the dread of syphilis. Serious varieties are *zoster hæmorrhagicus*, in which hæmorrhage occurs into the vesicles, and *zoster necrogenica* in which the skin sloughs, and is followed by scarring or keloid. *Diagnosis.* Herpes may be distinguished from all other vesicular conditions by the occurrence of the vesicles in *clusters* or constellations and their erythematous base. In regard to *prognosis*, herpes tends to spontaneous recovery in the course of a few weeks, except in the two grave forms above mentioned. The neuralgia which succeeds, however, is often very intractable, especially in the aged.

The *treatment* is quite simple. Protect the vesicles by starch or zinc powder, or paint with collodion, or use some soothing ointment. Quinine in large doses, 5 grs. 3 or 4 times a day, is reputed to be the best remedy for the neuralgia. For herpes preputialis give lead lotion.

IV. **Varicella,** the description and differential diagnosis of which has been dealt with (p. 591 and p. 601).

TABLE OF DIAGNOSIS.

Varicella.	*Smallpox.*
No symptoms before rash.	Three days before rash, sudden onset of illness with backache.
Soft pink papules becoming vesicular.	Shotty papules becoming vesicular or pustular.
Chest, neck and trunk, rarely face and limbs.	First on face and wrists, then trunk and legs in regular order.
Successive crops, and thus find small papules beside vesicles of various sizes.	**All one stage** (papular or vesicular or pustular) at one place.

V. **Scabies** is mostly a papular eruption (§ 464). But, in children, the vesicular element is apt to predominate, and it may then be mistaken for varicella. The burrows, and the marked tendency which scabies has to affect the soft skin at the bends of the joints and between the fingers should obviate such a mistake.

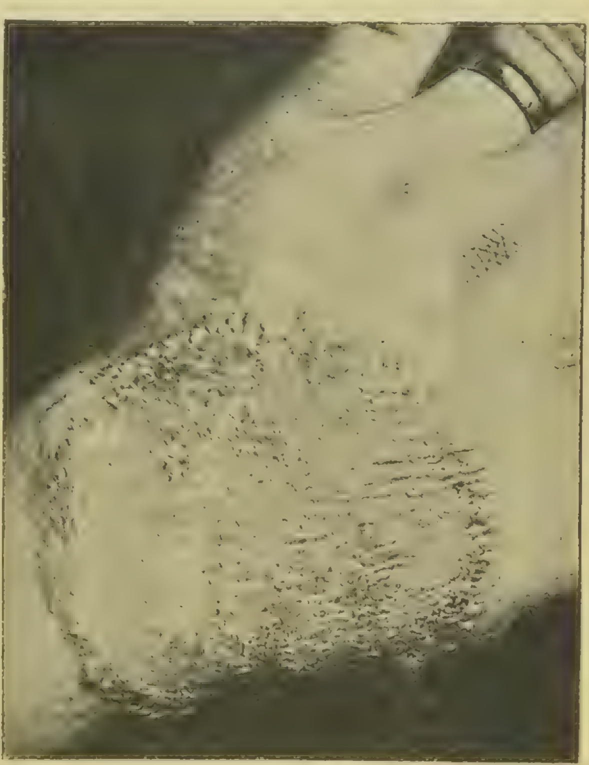

Fig. 136.—Hand of a woman suffering from TINEA CIRCINATA, in which the vesicular element is unusually prominent. A large ring of vesicles encloses a scurfy area. Verified by microscopic examination.

§ **479.** VI. **Tinea Circinata,** or ringworm of the body, is occasionally vesicular, especially when occurring on the wrists of adults, the arrangement of the vesicles in the form of a definite ring being so characteristic as to be unmistakable (Fig. 136). The usual naked-eye appearance of this lesion is a scurfy ring pale in the centre, red at the margin which with a lens is seen to be slightly raised with minute papules or vesicles. The favourite localities are the face, neck, and arms. When occurring in the genito-crural region it used to be (improperly) called **eczema marginatum.** On examining scrapings under the microscope, the mycelium (Fig. 137), and perhaps a few spores of the tricophyton tonsurans (the large-spored fungus, § 197) can be seen. The treatment consists of rubbing in ung. hyd. amm. chlor., or some other parasiticide.

§ **480.** VII. **Sudamina** are clear, scattered, non-inflammatory vesicles, like droplets of water, about the size of a pin's head, occurring in conditions such as acute rheumatism, which are attended by very profuse perspiration. They do not give rise to any inconvenience, and disappear in a few days. They are a non-inflammatory disorder of the sweat glands, whereas *miliaria* (commonly called prickly heat) are a mildly inflammatory condition of the same glands characterised by similar papules and vesicles on a red base.

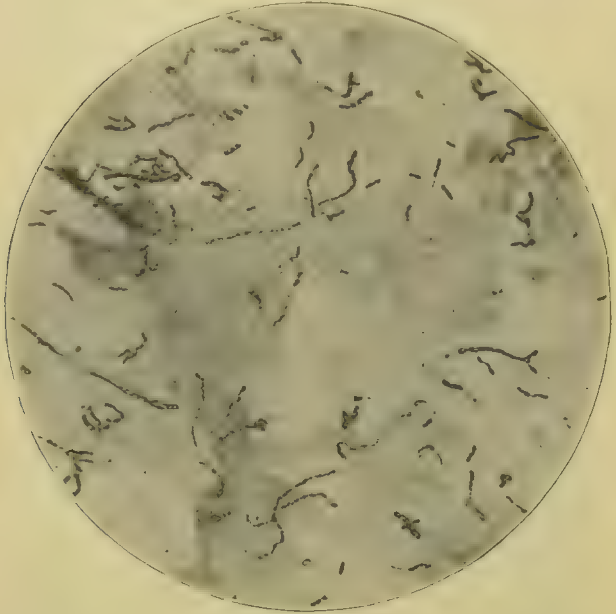

Fig. 137.—Mycelium of TINEA CIRCINATA (ringworm of the body). A scraping from the skin, stained by Gram's method. Mycelium of equal segments having truncated ends bifurcating in places; spores almost absent. Compare fig. p. 872.

VIII. **Hydrocystoma** is a rare disease, characterised by deep-seated, tense, translucent vesicles, occurring on the face. They vary in size from a pin-head to a pea, and are very persistent, lasting for months. They are formed by a cystic swelling of the duct of the sweat-gland; and never become purulent. They disappear spontaneously, chiefly in cold weather, and leave no trace. The disease occurs chiefly in middle aged women, especially in those whose life is spent in a warm, moist atmosphere, or who perspire much. It frequently comes on as warm weather sets in. The *treatment* consists in puncturing the vesicles.

§ **481.** IX. **Dermatitis Herpetiformis** is the term used by Duhring[1] for a large and widely varying group of eruptions which were formerly classed under pemphigus and various other headings.[2] Dermatitis herpetiformis may be defined as a relapsing disorder of prolonged duration, characterised by the appearance of successive crops of erythematous or papular elements, always in clusters, which usually go on to the formation of vesicles, pustules, or bullæ, are always attended by intense irritation, and sometimes by pigmentation. Different varieties are described according to the element which predominates. In some cases (the *erythematous* variety) the preponderating eruption consists of circumscribed patches of bright red erythematous or semi-urticarial inflammation, which spread by raised edges and leave a pigmented centre. In another, and perhaps a commoner variety (the *papulo-vesicular*), vesicles which vary from the size of a pin's head to a split pea predominate. Sometimes these vesicles become bullæ as big as a walnut (the *bullous* variety). They are always in clusters of two or three, and may coalesce; and sometimes the vesicles become pustular. Sometimes the predominating element is a papular one, and because of the itching the heads of these become scratched and covered with blood-stained scabs. Still more often these various elements are commingled, for the different lesions apparently represent different stages of the same pathological process. The intense itching is a very notable feature in all the varieties, especially the papulo-vesicular one; which probably corresponds with what was known as pemphigus pruriginosus. Scars and temporary pigmentation may ensue. In many cases the general health seems undisturbed, but often the appearance of each crop is attended by pyrexia, and occasionally gastro-intestinal disturbance. As regards its distribution, the elements have a tendency to be symmetrical and to favour the flexor surface of the wrists, the axillæ, groin, abdomen and buttocks. The mucous membrane of the mouth and pharynx may also be involved. Each successive crop lasts from one to four weeks; each attack being separated by longer or shorter intervals of comparative freedom. In this manner the disease may go on for months or years.

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[1] *American Journ. of the Medical Sciences*, Feb., 1891, and elsewhere; also New Sydenham Society's publications.

[2] Synonyms of Dermatitis Herpetiformis:—Pemphigus pruriginosus; pemphigus circinatus (Rayer); pemphigus composé (Devergie); herpes gestationis (Milton, 1872); herpes circinatus bullosus (Wilson); herpes phlyctenodes (Gilbert); erythema bullosum; dermatitis multiformis (Pillard); hydroa gestationis (Liveing); hydroa herpetiforme; hydroa bullosus; pemphigus arthritique (Bazin); Duhring's disease; impetigo herpetiformis of Hebra; dermatite polymorphe prurigineuse, ou douloureuse, chronique à poussées successives (Brocq).

Diagnosis. The disease differs from pemphigus vulgaris in the following respects:—(1) the smaller size of the vesicles or bullæ, which are (2) constantly arranged in clusters; (3) the presence of erythematous patches beneath the vesicles and elsewhere, and the multiform character of the eruption, and (4) the presence of itching. Nevertheless, it will be seen that all these differences are more questions of degree than of kind. From *eczema*, *urticaria bullosa*, and *erythema multiforme* the disease is distinguished by consideration of the above features.

Etiology. The disorder is more common in men than in women, and between the ages of 16 and 30. Many regard it as a dermato-neurosis, by reason of its symmetry and tendency to attack neurotic individuals.

Treatment. Arsenic is of great service and should be given in full doses. Quinine and salicin, and phenacetin for the irritation are useful. Locally, sedative lotions and ointments may be prescribed, much the same as those recommended in acute eczema (*q.v.*), while Duhring advocates sulphur ointments, 2 drachms to the oz., and tar lotions, *e.g.*, liquor carbonis detergens 5 drachms to the pint.

§ 482. X. **Pemphigus** is one of the rarer diseases of the skin, characterised by the presence of bullæ and constitutional symptoms of a mild or severe nature. P. *chronicus* or *vulgaris* is the more common and typical variety, in which the bullæ develop in crops, each bulla varying in size from a pea to a hen's egg, being tense with clear fluid, which becomes turbid, purulent and occasionally hæmorrhagic (p. hæmorrhagicus). The bulla is characterised by having no ring of erythema round its base. The fluid is either absorbed with formation of crusts, or the blebs burst leaving a raw surface on which new epidermis soon develops. Almost any part of the skin may be affected, as well as the mucous membrane of the mouth and nose. While each bulla only lasts a few days fresh crops may continue to come out for several months. The constitutional disturbance depends largely upon the number of bullæ and the frequency of the crops, the prognosis for recovery being good when the number is small and grave when abundant. Many cases are apt to recur throughout life. *Acute P.* is a rarer form found more in children than adults, and terminating in a few weeks either in recovery or death. In P. *foliaceus* the bullæ are very thin and flaccid and rupture early; but the epidermis instead of re-forming continues to peel off until large areas of red, raw, exuding surface are exposed, with epidermis folded at the margins—a point which distinguishes it from eczema rubrum. This process slowly extends for a year or two until the whole body may be involved, and a fatal issue ensues. P. *vegetans* is the rarest variety, beginning in the mouth, which develops papillomatous vegetations on the base of the bullæ and usually ends fatally in a few months.

The *Etiology* of P. is obscure. It is much more frequent in infancy, and is about twice as common in the male (Kaposi). At different times pemphigus has been associated with renal disease, anomalies of the female sexual organs, and lesions in the spinal cord or sympathetic system, but none of these are constant. Contagion has often been suspected, and Pernet and various other observers have found bacteria in the bullæ, but as Kaposi remarks, the etiological value of such findings is not established.

Treatment. The treatment of pemphigus is usually regarded as rather hopeless; but arsenic in gradually increasing doses is sometimes efficacious, and in some cases seems to have quite a specific action. Quinine and other tonics and a generous diet are called for, especially in cachectic cases. Among local remedies the continuous bath in severe cases, and dusting powders or boracic ointment to protect from septic influences, in milder cases, are the best.

XI. The description of the other diseases occasionally characterised by vesicles or bullæ—erythema multiforme, urticaria, drugs, congenital syphilis, leprosy, etc., will be found under their respective headings. Occasionally streptococcic dermatitis gives rise to large bullæ.

GROUP III. PUSTULAR ERUPTIONS.

Eruptions in which the elements are mainly pustular naturally fall into three classes.

a. Superficial Pustules.

I. Impetigo contagiosa.
II. Ecthyma.

b. Pustules on an Indurated Base.

III. Pustular syphilide.
IV. Sycosis.
V. Pustular acne.
VI. Pustular folliculitis.
VII. Bromide and other drug eruptions.
VIII. Variola.
IX. Acute glanders.
X. Pustular tuberculide.

c. Furuncular Eruptions with a Slough.

XI. Boils.
XII. Carbuncles.
XIII. Kerion.

Hebra wrote in 1870 "all pustules must be regarded as secondary morbid products, and hence are not fitted to form an independent series of cutaneous diseases." Curiously enough almost the only exception to this dogma is to be found in that rare condition Impetigo Herpetiformis, which was first described by Hebra. *Eczema and all the diseases mentioned* *in Group II. may become pustular*, owing as we now know to infection by pyogenic cocci. Conversely, nearly all the pustular diseases just mentioned may start as vesicles.

According to Dr. Sabouraud (*The Lancet*, Aug. 10, 1901, p. 403, and *B. M. J.*, Aug. 3, 1901), there are three prevailing cocci found in the skin—(1) the streptococcus of Fehleisen which is the cause of the impetigo contagiosa of Tilbury Fox, and for this he recommends 1 or 2 per cent. zinc sulphate; (2) staphylococcus aureus (Rosenbach), which invades the follicular orifices and causes pustules, being the cause of all primary and secondary pustular lesions, and for this he recommends sulphur 19 grms., alcohol 30 grms., aq. rosæ 100 grms.; and (3) staphylococcus griseus, which causes pityriasis simplex and seborrhœic dermatitis, and is probably the same as the morococcus of Unna. It is also called the staphylococcus cutis communis because it is the commonest of all. For this he recommends oil of cade 10 grms., yellow binoxide of mercury 1 grm., and pet. moll. grm. 30. All these three organisms, Dr. Sabouraud suggests, are probably polymorphic varieties of one and same microbe.

a. Superficial Pustules.

§ 483. I. **Impetigo Contagiosa** (syn. porrigo) is frequently met with on the faces of school children, and is so called because it is readily conveyed from one child to another. At first the spots are vesicular, but they readily become pustular. They vary in size, and are discrete, but may run together if near each other. In the course of a few days they dry into yellow crusts, which, falling off, leave a flat congested mark covered by new cuticle. They never scar unless scratched. The favourite positions are the face, especially round the mouth, scalp, and hands of children, but they may occur on any part of the body. If untreated, fresh pustules appear in other places for a few days to a week or so; or the disease may die out spontaneously in a few weeks. It is a trivial disorder, apart from the disfigurement, without constitutional disturbance, and with only slight itching. The disease may be conveyed by contagion to other parts of the same or to another individual.

Etiology. Impetigo will spread through a school or family of children, attacking weak and strong alike. Adults enjoy remarkable immunity, but occasionally an adult will contract it from the children. The essential cause is a pyogenic coccus (streptococcus and staphylococcus). Coccal infections of this class may give rise, as just mentioned (p. 854), to vesicular and vesico-pustular eruptions of several kinds.

Diagnosis. The impetigo pustules are readily distinguished from acne, sycosis, pustular syphilide, and all other pustular eruptions by (i.) their superficial character, and (ii.) their typical localities. Impetigo contagiosa is distinguished from eczema impetiginodes, by the absence of the eczematous element.

The *treatment* is extremely simple. The crusts must first be removed with warm water, a little sweet oil, or a starch poultice; and then a few applications of sublimate solution, or ung. hyd. am. chlor. are sufficient. Mercury is almost a specific in pustular affections.

ECZEMA IMPETIGINODES is really a pustular form of eczema as already described.

Impetigo Herpetiformis, or as it might be more properly called PYÆMIC IMPETIGO, is a rare disease decribed by Hebra, the differentiation of which from dermatitis herpetiformis is often difficult. It is a pyæmic process of the skin, characterised by the appearance of clusters of miliary pustules, usually starting on the inner surface of the thighs, whence they spread to almost the entire integument, generally associated with the pregnant or puerperal state, and terminating fatally. The tongue, palate, pharynx, and even the œsophagus have been the seat of pustules and superficial ulceration. There is considerable fever of pyæmic type, with delirium and vomiting, and each fresh crop of pustules is attended by rigors, and increasing prostration. All but two of the recorded cases have been connected with the later months of pregnancy; and if the patient recovers from the first attack the disease seems to be apt to recur in a subsequent pregnancy. Nearly all the recorded cases have terminated fatally, sooner or later.

The *treatment* should be conducted on the lines of other septicæmias (Chapter XV.). Vigorous local measures should be employed; probably the acid nitrate of mercury vigorously applied in the early stage might cut the process short. But when the disease is established, it would perhaps be better to make hourly applications of 1 in 1000 corrosive sublimate (after the manner of cancrum oris (§ 147). Any vaginal or uterine discharge should be attended to by frequent irrigations.

II. **Ecthyma** is a term which is gradually falling into disuse, but which is still sometimes used to describe *large isolated pustules*, or the superficial sores which form part of impetigo, scabies, pediculosis, etc., in children and aged persons broken down in health, and wanting in personal cleanliness. They may have to be distinguished from scabies in children and from suppurating syphilides.

b. Pustules on an Indurated Base.

§ 484. III. **Pustular Syphilides** are of two types:—1. *Small papulo-pustular syphilide* (acneform syphilide, or lichen syphiliticus pustulosus), consists of spots about the size of a pin's head, upon a hard base, which in a week or ten days scab off, leaving the characteristic indurated papules with depressed centres. They are arranged in groups, circles, or circular lines. 2. *Large pustular syphilide* (rupia, ecthymatous syphilide, variola syphilitica), consists of pustules varying in size from a split pea to a halfpenny, flat or hemispherical, and surrounded by a raised brick-red infiltrated margin. They may be grouped, ringed or isolated. The pustule bursts, the pus escapes, and crusts are formed with ulceration beneath them. The ulceration tends to spread serpiginously, and leaves permanent scars, rings and pigmentation. Both varieties may occur on any part of the body, and both indicate a malignant type of syphilis. The smaller pustular syphilide may have to be *diagnosed* from *acne* by the presence of comedones, and slower course in the latter. When on the face pustular syphilide may be hard to distinguish from *lupus vulgaris*, but the youth of the patient in the latter complaint, and the extremely slow rate of progress, may aid us. When pustular syphilide is diffuse it may be mistaken for *variola*, but in the latter there is a history of a vesicular stage, of backache and constitutional symptoms.

§ 485. IV. **Sycosis** is a term applied to a slowly growing papulo-pustular eruption affecting the sebaceous glands and hair follicles of the beard and sometimes the moustache. Indurated papules at first appear and some of these undergo suppuration. The disease is usually contracted at the barber's shop, but grooms also take it from horses and other animals suffering from ringworm. Clinically there are three conditions which most people admit may present the appearance which we describe as sycosis (συκον = a fig). (1) True sycosis (syn. folliculitis barbae) in which the pustules become large and indurated, and in this later stage the hairs can be easily drawn out with their macerated and softened sheaths entire. This is due to staphylococci, and on that account is sometimes called coccygenic sycosis; and is usually contracted at the barber's. (2) Tinea sycosis (syn. hyphogenic sycosis) is ringworm of the beard due to the trichophyton tonsurans. The large spored ringworm in children, and the ringworm of horses, cows, cats and dogs may produce this disease. There are two varieties, (a) *superficial*, characterised by scaly red rings, in which the hairs are only slightly involved, and (b) *deep-seated*, in which hard nodules and lumps are formed with suppurating hairs. In this form the hairs are easily pulled out from the onset. (3) *Eczema barbæ* taking on suppuration consists primarily of a general inflammation of the skin with pustules around some of the hair follicles secondarily. In true sycosis the pustules are the predominating element, and the intervening inflammation is secondary. In eczema barbæ the eczematous condition affects the intervening parts and spreads on to the face as well. All three conditions may if untreated last for a considerable time.

It is sometimes very difficult to *diagnose* which of the three conditions named we have to deal with; unless the tricophyton be found. In the eczematous condition we can sometimes trace the origin to seborrhœa of the scalp. In *impetigo contagiosa* the pustules and crusts are quite superficial and readily distinguished from sycosis. The *treatment* is generally a matter of months. Salicylic and carbolic acids and mercury are in my experience the best reagents to use. Some advise epilation, but when the condition is markedly pustular it is an advantage to squeeze out the pus and pack the hole with ointment. The hair should be kept short. The eczematous variety is treated like other eczemas (§ 477).

§ **486**. Various other pustular eruptions may be mentioned.

V. **Pustular acne** is recognised at once by the presence of comedones, papules, and pustules on the face and sometimes the upper part of the back. This and the severe pustular form known as *acne varioliformis* is described in § 462.

VI. **Pustular folliculitis** is a papulo-pustular condition specially affecting the hair follicles, due to a pyogenic infection (staphylococcus aureus, according to Sabouraud). It is independent of eczema and affects only the hairy parts, especially the legs of male subjects. It may be distinguished from a syphilide by the fact that each papule or pustule involves a single hair follicle.

VII. **Iodides** and **bromides** sometimes produce pustular eruptions on a congested indurated base. Antimony, aconite, arsenic, sulphide of calcium, nitric and salicylic acids also produce pustular eruptions.

VIII. **Variola** (Small-pox). The concluding stage of the eruption in this infectious fever is another illustration of pustules forming upon an indurated base (§ 356).

IX. The eruption of **acute glanders** when it has reached a pustular stage is so much like small-pox that it may very pardonably be mistaken for it (§ 363).

X. A pustular **tuberculide** is described by some observers as a rare manifestation of tuberculosis, and is generally associated with other tuberculous signs. Occurring as papulo-pustules up to the size of a pea, they are distributed discretely or in groups over the face, the body, and the limbs.

c. Furuncular, or Sloughing Eruptions: viz., boil, carbuncle, and kerion.

§ **487**. XI. **A Furuncle**, or boil, is an acute, circumscribed, suppurative inflammation in the skin, varying in size from a small pin's head to a bean. When the process fails to pierce the skin it is called a *blind* boil. As a rule, however, the inflammation involves the surface, which breaks and permits, in the course of a few days, of the discharge of the central necrosed portion, which is spoken of as the core. The cup-shaped cavity which is left heals by granulation, and a scar remains. The pain is considerable, especially in the early stages, and varies with the tension of the part. A specially severe form of boil is the "cadaveric boil," which is due to infection during a post-mortem examination. *Furunculosis* is the term applied to the condition in which boils are constantly recurring over a prolonged period, at different parts of the body.

Treatment. To protect the part from external irritation is the most important preliminary. Frequent ablution is necessary. Hot carbolic compresses relieve the pain and hasten recovery, and a weak tar ointment may be employed. Calcium sulphide is recommended internally, but I have not found it of much use. Iron and strychnine are useful, and the general nutrition should be improved and stimulants administered.

XII. **A Carbuncle** may be regarded as a combination of several boils side by side, constituting an inflammatory area spreading beneath the skin of considerable size, with numerous openings in the skin through which the pus pours, and a leathery slough forms as it were a sheet in the deeper layers of the derma. Its commonest position is the nucha or back, but it may occur on the sacral region from pressure, on the face (when a neurotic element can generally be traced), or elsewhere. The patient is nearly always advanced in years and is often lowered in health in some way. The pain and constitutional disturbance are often very severe, and, if the carbuncle be extensive, general pyæmia may ensue, or death from exhaustion. The *diagnosis* is never difficult, on account of the characteristic red, infiltrated, swollen, circumscribed area in one of the positions named, and honey-combed appearance with pus-discharging holes. The *causes* are much the same as those of boils, though carbuncle is more often the result of debilitated states, and diabetes, and is more often found in the aged suffering from cardio-vascular disease.

Treatment. In the early stage an ice-bag has been recommended, to check the inflammatory process, but I never saw any good done by this means. Warmth is, in my experience, far more useful to check the spreading, or, if this is impossible, to promote suppuration, and the separation of the slough. When, by unmistakable fluctuation, we know that suppuration has ensued, free crucial incisions should be made, the slough cut away as freely as possible, and frequent syringings every hour or so with a carbolic lotion, 1 in 100, adopted. Iron, arsenic, strychnine, and a liberal diet, should be freely administered.

XIII. **Kerion** is a condition described by Celsus, and occurs chiefly on the heads of children suffering from neglected ringworm. Superficially it resembles a carbuncle, but without the same induration. It is a circular, raised, inflamed, boggy area of skin through which are a number of pus-discharging holes. It is commonest on the scalp and beard, and is due to the large spored ringworm (see § 497).

GROUP IV. MULTIFORM ERUPTIONS.

Multiform eruptions are sometimes found in the following conditions—syphilis, scabies, eczema, erythema multiforme, varicella, leprosy, and dermatitis herpetiformis.

§ 488. General Characters of Syphilitic Eruptions. Syphilitic eruptions have already been referred to under syphilis (§ 405) and under papular and scaly eruptions. (1) They are of many different *kinds*, and several kinds may be present at one time (polymorphism). All kinds of elementary lesions may appear on the skin with the single exception of vesicles: eczema and other vesicular lesions are never found as a result of syphilis, a diagnostic feature of great importance. (2) The syphilitic *papule* may be regarded as a prototype of a syphilitic skin lesion; it is the starting point of them all. (3) The *features common* to all syphilitic rashes are their reddish-brown colour, generalised or symmetrical distribution, grouping in segments of circles, preference for the forehead and flexor aspects, polymorphism and absence of itching. The later skin lesions in malignant cases (in which a so-called tertiary stage occurs) differ however in being asymmetrical, and with a marked tendency to ulceration.

The clinical features which distinguish syphilides are explained by three histological facts. (1) All syphilides are due to a deposit in the dermis or epidermis of a cellular *infiltration*. Hence the colour does not disappear on pressure, and is followed by staining. (2) The cells constituting this gummatous or granulomatous infiltration are of low vitality. They do not organise into connective tissue, but tend to undergo either *involution* by absorption on the one hand or *suppuration* and pustulation on the other. Hence the depressed cup-shaped centre, and the great tendency to polymorphism. Hence also the absence of vesiculation or an eczematous form of eruption. (3) The infiltration *spreads centrifugally*. Hence the raised peripheral edge is the newest part, the shape most frequently assumed being that of a crescent, circle, or segment of a circle leaving a stained centre where the papule began. If these three principles be appreciated all the clinical features are explained.

Scabies (§ 464) as it occurs in children is nearly always a multiform eruption, consisting of papules, vesicles, sometimes pustules, wheals, scratch-marks and burrows. By the presence of the latter and the position of the eruption the diagnosis is arrived at.

GROUP V. NODULAR ERUPTIONS AND TUMOURS OF THE SKIN.

A nodule may be defined as a solid deposit in the skin, which is larger than a papule. The *commoner forms* are—I. lupus vulgaris; II. syphilitic gummata; III. various benign tumours (*e.g.*, sebaceous cyst, lipoma, rheumatic nodules, vascular nævi, etc.); and IV. epithelioma; while the *rarer forms* include—V. leprosy; VI. Bazin's disease; VII. molluscum contagiosum; VIII. molluscum fibrosum; IX. sarcoma cutis; X. actinomycosis; XI. yaws; XII. mycosis fungoides; and XIII. blastomycosis.

§ 489. I. **Lupus vulgaris** may be summarily defined as a chronic disease of the skin, characterised by a collection of reddish-brown, semi-translucent ("apple-jelly") nodules embedded in the corium, which give rise to some general thickening and desquamation, and have a tendency to ulcerate and to result in cicatricial atrophy. Their favourite position is the face, in which position the patches are rarely symmetrical, as in l. erythematosus. They occasionally affect the limbs—in 20 per cent. of the cases according to Kaposi.[1] The disease almost invariably starts early in life—in childhood.

TABLE OF DIAGNOSIS.

Nodular Syphilide.	*Lupus Vulgaris.*	*Lupus Erythematosus.*
Nodular or diffuse infiltration with raised edges.	"Apple-jelly" nodules in derma. Sebaceous follicles not specially involved.	Superficial erythema. Sebaceous follicles plugged with hard sebum.
Destroys more in a month than lupus in a year. Stellate scarring.	Destroys slowly and usually leaves puckered scar.	Never ulcerates, though may leave a superficial scar.
Sometimes symmetrical.	Asymmetrical.	Bat's-wing distribution on face. Generally symmetrical.
Adults.	First appears in childhood.	First appears in middle life.
Amenable to Hg and KI.	Hg and KI do harm.	Hg and KI no good.

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[1] "Lectures on Diseases of the Skin," p. 543.

The *prognosis* of lupus vulgaris turns principally on three things: (1) its position, (2) its extent, and (3) the general condition of the patient. Untreated, the disease will spread for years.

Treatment. General hygienic and tonic measures as for phthisis are useful, *e.g.*, good food, fresh air, malt and cod-liver oil. Local treatment consists in—(1) excision, which is only applicable to a very circumscribed area of disease; (2) curetting, followed by caustics, the result of which is often very satisfactory for a short time but unfortunately requires periodic repetition; (3) Finsen lamp (photo-therapy), which is best suited to limited patches and yields excellent results; (4) X-rays (radio-therapy), which are of more universal application, being easily applied to both small and large areas of ulceration, as well as to patches in the buccal, nasal and mucous membranes; (5) high frequency currents add to the good effects of (3) and (4); (6) hypodermic injections of tuberculin have produced good effects, and are particularly suitable for lupus scattered widely over the body; (7) caustics and escharotics like acid nitrate of mercury carefully applied, carbolic and salicylic acid, etc., are extremely useful to arrest, and

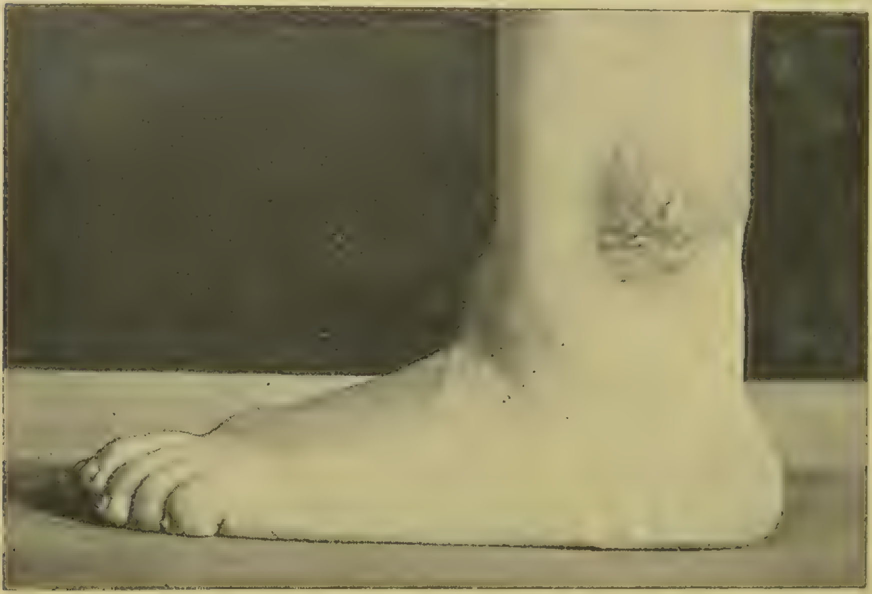

FIG. 138.—GUMMA OF THE ANKLE in a woman 45 years of age.

even, by prolonged treatment, to cure the disease when other methods are not available.

II. **Syphilitic Gummata** occur in the skin for the most part in the later stages of the disease. They are met with as round or ovoid nodules in or beneath the deeper layers of the skin. In the course of a few weeks they usually make their way to the surface in the form of an indolent abscess, which leaves a circumscribed punched out ulcer, sometimes of considerable depth. They may occur anywhere, but especially on the legs (Fig. 138), brow and sterno-clavicular region. They should not be lanced.

III. There are several other relatively common **benign tumours** or nodules originating in the subcutaneous tissue, which may involve the skin, *e.g.*, sebaceous cyst; fatty tumour; rheumatic nodules; fibro-neuroma; subcutaneous nævi; and lymphangiectasis. **Sebaceous Cyst** (syns. steatoma; wen) is a tense, rounded, painless cystic tumour due to the occlusion of a sebaceous follicle; sometimes associated with acne or a history of acne. They are usually single. For their eradication the *capsule* must be entirely removed or destroyed. **Fatty Tumours** are known by their doughy feel, lobulation, the puckered depressions seen on trying to lift up the skin over them. **Rheumatic Nodules** occur in successive crops, as small, hard or elastic nodules, sometimes adherent to the skin, usually freely movable beneath, sometimes tender on pressure. Their favourite situation is over the fibrous tissue of the superficial bones, that is to say, chiefly around the joints and along the spine.

IV. **Epithelioma,** epithelial cancer, affects the skin in three forms, and the favourite seat of all three is the face. 1. In the *papular* form it is found as hard, glistening, pale, flat papules, which grow very slowly, become cracked, fissured, and ulcerated (*vide* ulceration). 2. The *nodular* or deep-seated form is less frequently met with. It occurs as close-set flat or slightly raised, "very firm and somewhat translucent nodules. In the course of months or years it grows into a spherical or flat, hard tumour, whose surface is shining, waxy or rosy, traversed by vessels, irregularly nodular. As the result of spontaneous retraction the centre is often drawn in like an umbilicus; the edges are steep and smooth."[1] Later, ulceration occurs. 3. Papillomatous or warty growths (*malignant papillomata*) are occasionally met with. All three may be found in the same individual, but the first is the most common and the slowest to grow. The favourite sites are the lower lip—at least 50 per cent.—the tongue, and external genitalia. The majority of cases occur in men. Lesions having these features occurring in a person past middle life should always be examined microscopically, and steps taken for their eradication.

§ **490.** Certain **rarer forms** of nodule and neoplasm also affect the skin.

V. **Leprosy** (syn. Lepra, elephantiasis Græcorum, leontiasis satyriasis) is a chronic constitutional disease, characterised by pigmentatory, sensory and nodular changes in the skin, due to a specific microbe affecting the skin and nerves. Leprosy used to be a widely prevalent disease, but only imported cases are now found in England. It is still endemic in Norway, Sweden, parts of Russia, Turkey and the Turkish provinces, and in China, India, West Indies, etc. Owing to its distribution along the coasts of [illegible], Mr. Jonathan Hutchinson believes that the infection is conveyed by fish. It is communicable from man to man, though its infectivity is feeble, and probably only through an abrasion of the surface after the manner of syphilis and tubercle. From time immemorial lepers have been avoided and isolated, formerly because of the terrible aspect of the disease, latterly because of its infectivity. The disease is met with in two clinical forms in its earlier stages. a. *Maculo-anæsthetic* leprosy, which consists of patches of anæsthesia, sometimes of pigmentation or leucodermia, usually associated with thickening of the nerve trunk connected with the part, and a wide-spread eruption of reddish spots and patches over the body. b. *Nodular* leprosy, in which are found small diffuse thickenings without much alteration of colour of the dermal tissue and mucous membranes. These increase to form bosses, and occurring on the face, give to the patient a leonine aspect in course of time (facies leonis). The viscera are similarly involved, and wherever the granulomatous material is formed the characteristic bacillus is found, which has a very close resemblance to the bacillus of tubercle, *Mixed* forms of these two types are occasionally met with. The course of the disease is extremely prolonged and generally fatal. Any age may be affected. It is infectious as above mentioned, and is endemic in certain countries where want of cleanliness and hygienic principles lead to the transmission of the disease from person to person.

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[1] Kaposi, "Diseases of the Skin," 1895.

Treatment.—Choulmougra oil, ♏200 to 300 in capsules per diem, has certainly arrested the progress in some cases, when given in gradually increasing doses.[1] Hypodermics of the same remedy or of mercury, as in the treatment of syphilis (*q.v.*) have controlled the disease in certain cases.

VI. **Erythema Induratum Scrofulosorum** (Bazin's disease) is a rare tuberculous condition, affecting chiefly young strumous women, and characterised by chronic subcutaneous nodules in the calves of the legs, which may ulcerate. They are sometimes difficult to distinguish from syphilitic gummata, but the latter are much more rapid in their progress. The treatment is by rest in a horizontal posture, constitutional treatment, and the application of a Martin's rubber bandage.

A *spurious form of Bazin's disease* is met with in young persons who have feeble circulations with a tendency to cyanosis. This leads to hypostasis of the extremities, especially of the legs, the skin of which is apt to be thickened and livid at certain spots, but the subcutaneous nodules of Bazin's disease are wanting.

VII. **Molluscum Contagiosum** consists of rounded semi-globular, pearl-like elevations, of whitish or pinkish colour, varying in size from a pin's head to a pea, and semi-translucent appearance. A tiny depression is found in the centre through which the contents can be squeezed. If left alone, inflammation and suppuration may occur, with spontaneous cure. The treatment consists either in snipping them off or in squeezing out the contents and touching the bases with silver nitrate or iodine.

VIII. **Molluscum Fibrosum** is a rare condition which consists in the formation of fibrous tissue in the deeper layers of the corium, slowly developing into tumours of varying size (up to 32 lbs.), which may be sessile or pedunculated. Their favourite situation is the back. The *treatment* is their removal by knife or ligature. One case under my care improved by painting with ethylate of sodium.

IX. **Sarcoma Cuits** may affect the skin either as a primary affection or secondary to deposits elsewhere. It is met with in the form of purplish tumours of varying size and varying generalisation, of hard or spongy consistence, and a raised infiltrating margin. A small deposit with satellites around it is very characteristic. Sarcoma may develop on pigmented moles, a melanotic sarcoma being then reproduced elsewhere.

X. **Actinomycosis** is a rare chronic affection of the subcutaneous tissue, usually starting in the jaw and spreading thence to the skin of the face and neck. It is due to the ray fungus or Actinomyces, which gives rise to a hard, slow-growing tumour, going on to ulceration, with a thin sero-purulent discharge, containing *yellow granules in which the ray fungus can be found* (§ 623). In other cases the disease attacks the lungs, the digestive tract, or the liver, causing much constitutional disturbance. The fungus enters through eating diseased grain improperly cooked. The prognosis is favourable if the disease is on the surface where it can be dealt with surgically, or if taken early, when large doses of pot. iod. control the growth.

XI. **Frambœsia** or **Yaws** is a chronic disease endemic in the tropics, rarely attacking the white population. The commonest form of eruption has a fungoid or raspberry-like character, whence its name is derived. Some observers, like Mr. Jonathan Hutchinson, consider the disease as identical with syphilis, modified by the climate; but it has been successfully inoculated into syphilitic patients and is probably not identical with syphilis.

XII. **Mycosis Fungoides** is a rare condition, characterised by the formation, after a long preliminary period, of reddish fungoid tumours. In the preliminary stages, which may last for months or years, there is an erythema or a scaly eczema attended by a good deal of itching, followed by brownish-red papules which leave pigmented and atrophied depressions, and are finally followed by smooth purple tumours, sessile or pedunculated, which gradually ulcerate. The eruption usually appears on the trunk, and gradually leads to emaciation and death. Arsenic in full doses subcutaneously injected is said to control the disease to some extent, and X-rays have proved useful.

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[1] See a case recorded by the author, Clin. Soc. Trans., about 1896.

XIII. **Blastomycosis** is an extremely rare disease affecting chiefly the face and hands, characterised by papillomatous ulceration simulating tuberculosis cutis, and due to the fungus blastomyces.

GROUP VI. ULCERATION

§ 491. An ulcer is a loss of substance of the derma and epiderma, exposing a granular surface, which secretes a sero-purulent fluid. Ulcers must not be confused with large vesicular or bullous lesions, such as occur in pemphigus foliaceus, in which the skin is only denuded of its cuticle. For clinical purposes ulcers may be divided into 4 groups—

(a) *Idiopathic* or *inflammatory ulcers*, caused by injury, hypostatic congestion, or varicose veins, and often aggravated by some blood dyscrasia such as gout, anæmia, or scurvy.

In the *treatment* of chronic ulcers the main point to remember is their absence of tendency to repair. (1) Many *local applications* have been tried. If the discharge is watery and excessive, and the granulations turgid, astringents are called for, such as zinc sulphate, boric acid, or lead lotion, or the painting on of nitrate of silver, 20 grs. to the oz., or the use of the solid stick, which acts also as an excellent stimulant. Another good stimulant is ung. hyd. ox. rub., which acts in the same way. For the troublesome itching, carbolic lotion, 1 in 50 or 1 in 100, freely applied on lint or rag, often gives relief. Meat extracts and oxygen have been used as applications, and various protective dressings such as strapping and starch dressings are often successful. (2) Rest in the horizontal position is more efficacious than any other kind of treatment for ulcer of the leg, because the deficient return of the blood is one of the factors which prevent repair. With the same object a Martin's rubber bandage, a flannel roller, an elastic stocking, or a Scott's dressing is advantageous. (3) A liberal diet, and a moderate use of stimulants are often successful, combined with tonics, and among the internal remedies which I have found successful with aged persons is tr. opii, ♏ 2 to 5, thrice daily. It acts by improving the tone of the cutaneous vessels, and promotes repair. It may be combined with strychnine, which is one of the best tonics for the aged with which I am acquainted. (4) Incisions may be made through callous, indolent edges, either at right angles or parallel, to release the adhesions between them and the deep parts. Grafting by Tiersch's or some other method is, however, more successful.

(b) The *contagious ulcers* are hard chancre, soft chancre, the ulcer at the seat of inoculation of glanders, and certain tropical ulcers due to the poisonous stings of certain insects, and the inoculation of certain tropical diseases.

(c) *Neuropathic ulcers, e.g.*, perforating ulcer in tabes dorsalis.

(d) *Infiltrating* or *neoplastic ulcers* are due to the breaking down of some infiltration which has invaded the skin or subcutaneous tissue, and which can be detected in the tissue around such as syphilis (rupial ulceration and breaking down of a gumma), lupus vulgaris, tuberculosis of the glands breaking down (strumous ulceration), epithelioma, rodent ulcer, and leprosy.

The differentiation of these several varieties depends largely on the history, the associated symptoms, the distribution and character of the ulcers, and these have for the most part been given under their respective titles.

An **infiltrating, ulcerating,** *and* **scarring** *eruption in a person of young or middle age is practically either* syphilis, lupus, *or* tuberculous ulceration. *If it occurs over* 40 *or* 45, epithelioma *and* rodent ulcer *enter into the category.*

I. **Syphilitic Ulceration**—other than the primary chancre—is of two kinds—(1) the breaking down of a large papular or lenticular syphilide *in* the skin gives rise to shallow irregular ulceration which may be covered with a scab which resembles the layers of an oyster shell (**rupia** of older authors). (2) The breaking down of a gummatous nodule which has started *beneath* the skin produces a deep punched-out ulcer. The three characteristic signs about all syphilitic ulcerations are—(1) the peripheral ring of infiltration, (2) the punched-out edge, and (3) the comparatively rapid march. The diagnosis from lupus and rodent ulceration is given in tabular form below.

TABLE OF DIAGNOSIS.

Syphilitic, rupial, or gummatous ulceration.	*Ulcerating lupus.*	*Rodent ulcer.*
Anywhere.	Chiefly face.	Chiefly face.
Adult life.	Begins between 10 and 20 years.	Over 40.
Progress rapid, destroying in weeks what others do in months.	Very slow.	Slow.
Sharp, clear-cut, punched-out, deep.	Edge rounded, sloping and surrounded by apple-jelly nodules; superficial.	Edge rolled and *hard*; deep in later stages.
Discharge copious, offensive.	Scanty, inoffensive.	Scanty, and in later stages sanguineous.
KI and Hg efficacious.	No good.	No good.

NOTE.—A small piece may easily be removed for microscopic examination in any doubtful case.

II. **Lupus Vulgaris** may ulcerate, but only when near a mucous orifice or subjected to injury. The nodules around are sufficiently characteristic (§ 489).

III. **Tuberculous or Scrofulous Ulceration** may occasionally take its origin from tuberculous deposits in the skin, but more frequently spreads from a caseating gland or strumous bone disease. The edges of the ulcer are dark purple, and undermined, never rounded and clean-cut as in syphilitic ulceration, and there are generally evidences of the strumous diathesis, or scars from past disease of the same kind. The patients are usually children, occasionally an old person (senile struma).

IV. **Rodent Ulcer** is clinically to all intents and purposes a variety of epithelioma in which the loss of tissue is more pronounced than the new growth; is found chiefly on the upper half of the face after 40 years of age, and in the course of years will destroy the eyelids, eyeballs, and a great part of the face. It appears as a very shallow, roundish ulcer, with a flat base, scanty viscid secretion, and a very hard edge matted to the subjacent structures. It often cicatrises in the middle, so that the ulcer becomes crescentic. It is said to be more frequent in men. Its progress may extend over many years, but it may suddenly take on activity at any time; and it may, as the author has seen, take on the clinical and histological characters of epithelioma in the evolution of the case.

V. **Epithelioma** is apt in course of time to undergo ulceration, but the diagnosis rests upon the characters of the initial growth, which is always found in considerable quantity around when the stage of ulceration is reached. If a small piece can be examined under the microscope the typical "bird-nest" growths of epithelioma (which are absent in rodent ulcer) can be seen.

The treatment of *neoplastic ulcers* is of 4 kinds—(1) constitutional treatment appropriate to the nature of the lesion; (2) caustics and escharotics applied regularly and freely, so as to destroy the neoplasm around; (3) various operative procedures — removal, scarification, or cauterisation; and (4) the use of the X-rays or Finsen light. Excellent results have been obtained by the X-rays in the treatment of lupus, rodent ulcer and superficial epithelioma.

GROUP VII. WARTS AND EXCRESCENCES.

§ 492. This group, which does not include neoplastic formations referred to in the last two groups, consists of verruca (wart), condyloma, corns, papilloma lineare, acanthosis nigricans, porokeratosis, and angiokeratoma.

Verruca, wart, or papilloma cutis, is an excrescence consisting of thickened epidermis containing elongated papillæ. Warts may occur singly, or they may be multiple. They are most frequently met with on the hands. More rarely they occur on the head, face, or genital organs. The *cause* of warts is obscure, but they are undoubtedly in some cases contagious (Dr. J. F. Payne), and in that way spread over the hands and other parts of the body.

Varieties. 1. *V. Vulgaris* occurs on the hands, and forms a horny growth the size of a small pea. 2. *V. Plana*, a flat, dark brown elevation found on the face or back of old people.

3. *V. Acuminata*, which is moist, sessile, or pedunculated, usually quite small, but may grow to be as large as the fist, occurs chiefly on the genital organs, or where opposed surfaces are in contact, and resembles condylomata. 4. *V. Seborrhœica* is an oily, fawn-coloured, slightly elevated and rounded body, in which the papillæ are mixed with inspissated sebum. 5. *V. Necrogenica*, or "post-mortem wart," is a tuberculous infection of the skin which appears on the hands of doctors, post-mortem porters, leather-dressers, cooks, butchers, etc. It starts as a crimson, flat, indurated papule, which spreads, and sometimes becomes pustular, the pus drying and forming into a scab. A white and pink stellate cicatrix may be left behind as the disease progresses (Fig. 139).

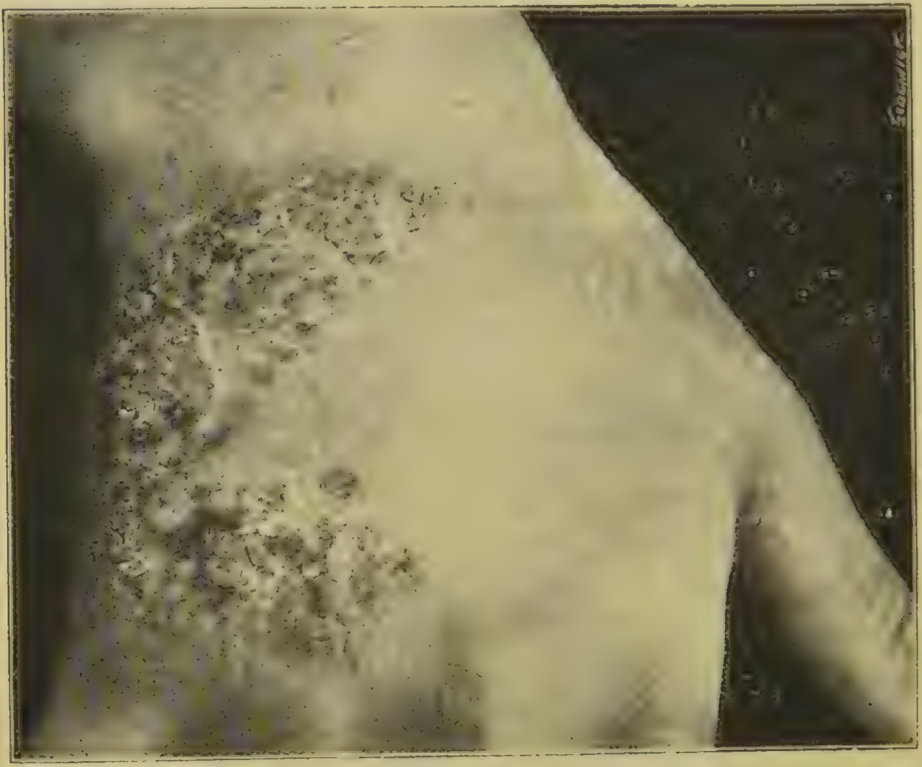

Fig. 139.—Verruca necrogenica on the hand of a gamekeeper, aged 35.

Treatment. Warts are best dealt with by applications of glacial acetic acid, potassa fusa or other caustics, or salicylic acid plaster. Small doses of mag. sulph. t.i.d. have cured some cases. V. Necrogenica is treated like lupus.

Syphilitic condyloma is really a papular syphilide occurring —1, on the mucous membranes; 2, near the junction of mucous membrane and skin; or 3, where opposed skin surfaces are in contact. They very commonly occur at the angles of the mouth, and between the buttocks or labia. They are slightly raised discs of various sizes (a pea to a shilling), covered with greyish epithelial or soddened epidermal flakes, and secrete a highly contagious fluid.

Corns are localised thickenings of the epidermis consequent on localised pressure. Thus the side of the toe is a common position. They may be cured by painting with salicylic acid (20 per cent.) and collodion every night for a week; soon afterwards the corn will flake off. *Soft corns* arise between the toes, due to hard corns becoming soddened with perspiration. Treatment consists in keeping them dry, with dusting powder, such as zinc oxide and starch, keeping the toes separate with small pads of cotton wool, and by relieving pressure.

Papilloma Lineare (syns. ichthyosis linearis; i. neuropathica; naevus unius lateralis; ichthyosis sebacea) (Fig., p. 844) is characterised by little warty growths crowded together in lines more or less parallel to the long axis of a limb, which appear to follow (but which do not really follow) the course or distribution of a nerve or vessel. The little warts and the excess of sebaceous matter pick up the dirt and produce a black appearance. The disease has been described under Ichthyosis Sebacea, § 475.

Acanthosis Nigricans is a rare condition characterised by progressive pigmentation of the skin, with papillary growths, terminating fatally in a few years. The colour of the skin varies from a sallow hue to bronze and dirty brown. It is generalised, but more pronounced in the flexures. The skin becomes thickened, and warty growths develop, chiefly in the flexures, anal, genito-crural, and abdominal regions. The disease may occur at any age after childhood. In most of the recorded cases it has been associated with abdominal cancer, but in others no cause has been found.

Porokeratosis, a very rare disease, occurring chiefly on the backs of the hands, is characterised by patches of atrophic skin, surrounded by a thin horny ridge or "wall" immediately inside which are seen tiny grey papules, which can be picked out. It is said to be a hyperkeratosis of the mouths of the sweat glands, with destruction of glands and hair follicles.

Angiokeratoma is a rare condition consisting of telangiectases, which develop into warty growths, occurring usually after chilblains, on the backs of the fingers, toes, hands, and feet. *Treatment* consists in employing warmth and electrolysis.

GROUP VIII. ATROPHIES AND SCARS.

§ 493. Scars, scleroderma, and atrophy of the skin may be considered together because they not only resemble each other clinically, but fibrosis of some of the cutaneous tissues and atrophy of others occur in varying degrees in all three conditions. The disorders met in this group are—

I. Scars. II. Atrophoderma. III. Scleroderma. IV. Keloid and its congeners Rhinoscleroma, Kraurosis Vulvæ, and Ainhum.

I. **Scars** may result from burns, wounds, or infiltrating or suppurating eruptions, in which there has been a loss of substance. If much deformity or loss of mobility results plastic operations are called for; but it is wonderful how much can be done in young patients by means of persevering massage with oleaginous substances, and especially, in my experience, cod-liver oil. Scars are liable occasionally to be affected by **keloid** (see below).

II. **Atrophoderma** (atrophy of the skin) occurs as—(a) Atrophy of the entire cutaneous covering is common in old age. (b) Lineæ albicantes is a term applied to the atrophic streaks found on the abdomen and breasts after pregnancy, over the hips and other parts when the patient has been getting rapidly stouter. A case of linear atrophoderma due to neuritis after enteric fever is recorded by Sir Dyce Duckworth.[1] The author and others have also observed cases which could be traced to a neuropathic cause. (c) *Unilateral atrophy* of the skin is met with in the condition known as Hemiatrophy Facialis (§ 612), which is of nerve origin.

III. **Scleroderma,** or fibrous thickening of the skin is met with in 3 clinical forms, all of which are more or less rare—(a) Localised (or morphœa); (b) diffuse; (c) S. neonatorum.

(a) LOCALISED SCLERODERMA (syn. morphœa of Erasmus Wilson; Addison's keloid) is a disease consisting of one or more localised ivory patches of sclerosed skin with, in the earlier stages, a congested lilac border. There are no subjective sensations but the tactile sensation is diminished. The tissues undergo slow contraction, and if some depth is involved it may result in a deep pit or furrow. On the other hand some cases undergo spontaneous resolution in course of years. The favourite situations are the face, neck, and beneath the breast. There is a tendency to symmetry, where more than one patch appears. It is thought that the shape and distribution of some patches corresponds with the distribution of a nerve, the supra-orbital being a common site, but it seems more probable to the author that the lesions are associated in some way with Head's sensory and visceral areas. Females are more prone to be affected in the proportion of three to one, and the disease appears mostly in the first half of life. Beyond the disfigurement and contraction the patient suffers but little inconvenience from this variety of the malady.

(b) DIFFUSE SCLERODERMA is a somewhat different affection to the foregoing, and consists of a parchment-like thickening and contraction of the skin, starting and predominating in the extremities. It progressively increases both in area and severity until the parts become completely hidebound and immobile. The face in such cases wears a smooth, expressionless aspect, which is readily identified. This disease being progressive is one of much gravity. By degrees fissures and ulcers form and some of the fingers may become gangrenous, and death from some intercurrent malady occurs. Many degrees of severity are, however, met with, and in some the condition only produces a constant liability to cold and to various superadded skin lesions.

(c) SCLERODERMA (syn. sclerema) NEONATORUM is a different disease. It appears congenitally, and is generally fatal in the course of a few weeks. The affected skin is bound down to the parts beneath, in red, tense, shining, symmetrical indurated patches.

The *treatment* of the 3 conditions is not very satisfactory. Local massage, especially with cod-liver oil may be recommended. Several slighter degrees of generalised scleroderma under my care improved under thyroid. The constant current has done good in the localised form.

IV. **Keloid** consists of a fibromatous deposit in the skin occurring primarily in unaffected skin, or secondly in old cicatrices. The lesion appears as a small firm nodule, of a crimson or pinkish colour (by which features it is readily distinguished from morphœa), which slowly, in the course of months or years, enlarges by means of tentacle-like processes thrown out into the surrounding tissues. At first it is raised above the skin level, but later, as the growth enlarges by spreading or the coalescence of several nodules, the centre becomes puckered and depressed. Such growths may remain stationary, spread, decrease, or ulcerate. If excised they immediately recur, and in that sense are malignant. Soothing applications are best. The negative pole of a mild constant current has been attended with favourable results.

Acne Keloid (syns. Dermatitis papillaris capillitii; sycosis capillitii) is a rare disease which occurs on the nape of the neck, and involves the margin of the hair. It is a slow pustular affection, resulting in keloid formation.

Rhinoscleroma is a chronic inflammatory affection characterised by the development of hard, circumscribed, nodular growths in the skin and mucous membrane, most commonly of the nose and nasopharynx, due it is believed to inoculation with a specific bacillus. The epidermis over them cracks, and crusts form over the fissures so caused. The bones and cartilage may be involved.

Kraurosis Vulvæ is an atrophy and contraction of the mucous membrane of the external genitals and adjacent perinæum of women. The labia minora and clitoris may almost disappear. The tissues are atrophied, with thickened patches, and may cause difficulty in parturition. Heitzmann reports a cure after curetting the thickened patches, and continuous applications of a 2 per cent. solution of salicylic acid, alternating with a similar solution of pyrogallol.

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[1] *Brit. Journ. Dermat.*, No. 62, vol. v.

Ainhum consists of a progressive, slow strangulation and amputation of one or more toes, by the growth of a narrow constricting band of hypertrophied skin, due to a parasite.

GROUP IX. PIGMENTARY AND VASCULAR ALTERATIONS.

§ **494.** Alterations of colour depend mainly upon the condition of the vessels and the amount of pigment in the skin. A *diminution* of pigment is not frequent, and occurs only in two conditions —(1) ALBINISM, a congenital condition in which there is deficient pigment in the skin and its appendages, and in the iris and choroid; and (2) LEUCODERMA (syn. vitiligo), a condition in which there is an absence of normal pigment in more or less circular areas which are surrounded by darker-coloured skin. The transition from the pale to the dark area is abrupt, and it is the dark-coloured margins which attract the notice of the patient. It may be congenital or acquired, and is probably neurotrophic in origin.

a. A *localised* increase of pigment occurs in :—

I. Chloasma.
II. Lentigo.
III. Pityriasis versicolor.
IV. Pigmentary and vascular moles.
V. Purpura.
VI. Urticaria pigmentosa.
VII. Xeroderma pigmentosa.
VIII. Xanthoma.
IX. Morphœa alba and nigra.
X. Leprosy.

b. A *generalised* increase of pigment occurs in :—(1) arsenical and silver pigmentation; (2) Addison's disease; (3) abdominal cancer; (4) cardio-vascular disease; (5) bronzed diabetes; (6) constipation; (7) melanotic sarcoma; and (8) acanthosis nigricans; but in these the pigmentation is subordinate to other symptoms.

I. **Chloasma** occurs in single or multiple patches of diffuse discoloration on various parts of the body, varying in shade from a light yellow to a deep brown. Several varieties may be referred to:—1. *Chloasma symptomatica* is met with most frequently in pregnancy or uterine disease, and its most usual position is on the face and round the nipples. 2. *Chloasma cachecticorum* occurs in association with malaria, cancer, senile atrophy, rheumatoid arthritis, abdominal tubercle or cancer, and exophthalmic goître. 3. *Chloasma traumatica* is the pigmentation beneath the garters, or around the waist in tight-lacing women, in pediculosis or scratching, and after sinapisms, blisters, etc. In this category may be included the pigmentation which follows chronic eczema, syphilis, psoriasis, or any other long continued afflux in the skin capillaries. 4. *Chloasma Caloricum* is the pigmentation due to sun and wind, or to heat, as on the shins of women who sit over the fire.

II. **Lentigo** (syns. Freckles, Ephelides). Freckles are multiple, circumscribed pigment spots on the portions of the body exposed to light. In advanced age they may occur anywhere, and are apt to become malignant by taking on an epitheliomatous growth.

III. **Tinea Versicolor** (syn. Pityriasis versicolor) is a vegetable parasitic affection of the skin, which appears as variously sized, irregularly shaped, dry, highly furfuraceous patches, yellowish in colour, found generally upon the trunk, and especially in the hollow of the breast bone, due to a specific parasitic fungus, the microsporon furfur (Fig. 140).

IV. **Pigmented and Vascular Moles** are distinguished from other pigmented conditions by their being congenital and being more or less raised. Nævus spilus is a smooth discoloured spot of otherwise healthy skin. Nævus verrucosus is rough and sometimes bristling with hairs (nævus pilosus). *Vascular moles* are purple spots of increased vascularity, usually a little raised above the surface, varying widely in size and thickness. They are sometimes stationary, but more often gradually increase in size. *Telangiectasis* is a term indicating a localised dilatation of the vessels of the skin, and is a frequent accompaniment of acne rosacea and other skin affections. Small spots are met with independently of any skin affection on the face and various parts of the body in healthy persons, particularly as life advances, or when the peripheral circulation is feeble. *Port-wine mark* is a venous and capillary dilatation over an area of skin.

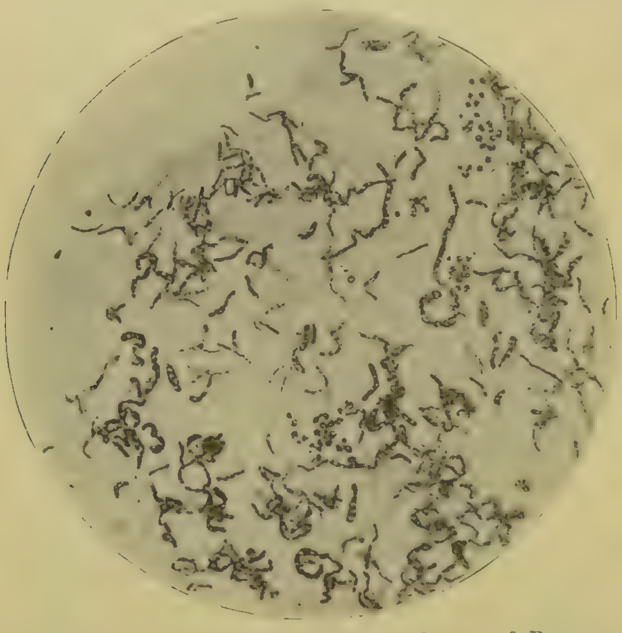

Fig. 140.—MICROSPORON FURFUR, the fungus of PITYRIASIS VERSICOLOR, × about 50. Shows the branching irregular mycelium and the constellations of spores. Stained by Gram's method.

Treatment. Leucoderma, lentigo, and chloasma are best treated with strong mercurial lotions (½ to 1 per cent.), but they are difficult to cure. For pityriasis versicolor thorough cleansing with a hard brush and soap is the essential treatment, together with ung. sulph. or a lotion of sod. hyposulph. (ʒj. to the oz.). Electrolysis, escharotics or excision give the best results in pigmented and vascular nævi.

§ 495. V. **Purpura** consists of dark, abrupt-edged purple spots due to extravasations of blood into the skin. The eruption does not fade on pressure. It is sometimes accompanied by similar extravasations into the mucous membranes and internal organs and hæmorrhages from the mucous surfaces. The constitutional symptoms vary considerably, and may be absent. Pyrexia, usually slight, occurs in about half the cases.

The *causes* of purpura are but little understood, but may be grouped under three headings :—1. *Local* and *mechanical* causes, such as *heart* *disease* and *old age*. The most frequent illustration of this is met with in eczema of the leg (indeed, most eruptions on the legs of old people) which assume a purpuric character on account of the hypostasis of the blood. Mitral disease is associated not infrequently with purpuric eruptions on the legs, especially if the patient walks about. 2. Purpura is associated with certain *nervous* conditions, such as cerebro-spinal meningitis (almost always), tabes, myelitis, hysteria, and, according to some, neuralgia. 3. *Changes in the blood* produce purpura in (i.) various *fevers*, especially typhus (typhus has been called purpura contagiosa) and cerebro-spinal fever, in which a purpuric eruption is constant; measles sometimes; malignant endocarditis and pyæmia occasionally; the initial stages of small-pox and in the *malignant type* of that and most of the other acute specific fevers; (ii.) certain *drugs* occasionally, especially mercury and iodide, quinine, copaiva, belladonna, ergot, salicylic acid, and the mineral acids; (iii.) *autotoxic* conditions, such as Bright's disease, hepatic disease, including cirrhosis, acute atrophy, carcinoma, and any aggravated jaundice; bad food also seems in some cases to account for the onset of purpura; and (iv.) *constitutional* diseases, such as leukæmia, scurvy, lymphadenoma, and great debility from any cause.

Four special *varieties* of purpura are recognised. (a) *Purpura Simplex* (morbus maculosus of Werlhof) is the name given to a mild attack of purpura for which no cause can be assigned. It is usually met with in young persons, and runs a benign course. (b) *P. Hæmorrhagica* is a severer variety, which may start as P. Simplex or independently. The spots are larger, and sometimes raised or œdematous. Hæmorrhage occurs from the mucous membranes; and pyrexia is more marked. (c) *P. Rheumatica* (Peliosis Rheumatica, Schönlein's disease), and *Henoch's Purpura*, both of which are described elsewhere. (d) Purpura and erythema are related, the first being due to an exudation of all the constituents of the blood, the second to an exudation of serum. Consequently it is not surprising to find that many intervening stages of the two are met with, and are known as *purpuric erythema*.

The *diagnosis* of purpura is easy, but difficulty lies in ascertaining its cause. The differentiation of *scurvy* has been given (§ 411). To ascertain the cause carefully consider the previous history and thoroughly examine every organ. If no cause can be found the disease is called P. idiopathica, simplex or hæmorrhagica.

The *prognosis* is extremely grave when associated with the specific fevers, or with a high temperature. P. simplex usually results in recovery in a few weeks; p. rheumatica is rarely fatal, though it may last for months or years and may recur; Henoch's purpura is favourable as regards recovery, but is apt to recur.

The *treatment* is unsatisfactory; arsenic, iron, ergot, and ol. terebinth deserve trial. A thorough inquiry should always be made as to the causes above mentioned.

VI. **Urticaria Pigmentosa** (described by Nettleship in 1869 and so named subsequently by Sangster) is a chronic or recurrent urticaria, in which each crop of wheals leaves behind spots of brown pigmentation of considerable persistency. The disease starts in early childhood; it may cease spontaneously about puberty, but it more frequently lasts for many years. The *treatment* is like that of urticaria, plus calcium chloride and bromides.

VII. **Xeroderma Pigmentosa** (syn. Kaposi's disease) is a very rare disease of a chronic progressive character starting in early childhood, often in members of the same family, and marked by small dark freckles with atrophy and contraction of the skin between them, and the occurrence of telangiectases. There is also a distinct tendency to a malignant new growth, both in the skin and the internal organs in the shape of a malignant sarcomatous or cancerous infiltration. The distribution is universal, and the contraction gives rise to eversion of the eyelids and other orifices. It usually terminates in death before the age of 22.

VIII. **Xanthoma** (syn. Xanthelasma) is a rare condition most commonly occurring in the face in cases of diabetes and chronic jaundice. It consists of yellowish nodular deposits in and beneath the cutis, of a spherical or ovoid outline, varying in size from a millet seed to a bean, or larger. A case in which the hands were involved is recorded and figured by Dr. Phineas Abraham.[1]

IX. **Morphœa Nigra** and **Morphœa Alba** are words employed by Erasmus Wilson for the disease, which following the Germans, we now describe as localised scleroderma (§ 493) when attended by excess or deficiency of pigment.

X. **Leprosy** (§ 490). Patches of pigment and white spots may occur in the early stage of anæsthetic leprosy; and dark spots occur, especially on the face, in the early stage of nodular leprosy.

GROUP X. DISORDERS OF THE SWEAT.

§ **496.** Four disorders of sweat are met with, anidrosis, hyperidrosis, bromidrosis, and chromidrosis.

I. **Anidrosis** is not common apart from the conditions mentioned in Group VIII.

II. **Hyperidrosis** is the term applied to an excessive secretion of the perspiration, and may be general or localised. When general it may be due to a lowered neuro-vascular tone, excitement, corpulency, or the use of stimulating foods and drinks. It also occurs in fevers at the crisis, in ague, acute rheumatism, chronic tuberculosis. The *localised* form affects most often the feet, axillæ, and palms. The sweat mixed with sebaceous secretion decomposes on the clothing, and gives rise to a pungent and disagreeable odour. The feet are apt to become tender, and eczema may supervene.

The *treatment* of hyperidrosis consists of the application locally of a mixture of tr. belladonna and water equal parts, or sponging with vinegar and water or quinine 1 per cent. in Sp. V. R. are useful, and internally hypodermics of atropin, or the administration of arsenic, and the mineral acids or tonics. *Local* hyperidrosis, especially of the feet, is troublesome to get rid of. The stockings should be changed several times a day, and put into a saturated solution of boracic acid before being used again. Dusting powders, or a lotion of lead, calamine or zinc, containing methylated spirit, may relieve the slighter forms. Applications of tannic acid, chromic acid, salicylic acid, in powders or pigments, and of diachylon plaster, should all be tried.

III. **Bromidrosis** is the term applied to a disturbance in the function of the sweat glands in which the perspiration has an offensive odour. Mere excess of perspiration in the feet or axillæ may render the person disagreeable to his companions. The subject is therefore interwoven with hyperidrosis, to which reference should be made for the treatment.

IV. **Chromidrosis** is a rare disturbance in the function of the sweat glands in which the perspiration is coloured. It is probably due to indican, which after being excreted by the sweat glands, is acted upon by the air and coloured blue or black.

GROUP XI. DISEASES OF THE SCALP AND HAIR.

§ **497.** The diseases special to the scalp and hair are:—

I. Ringworm.
II. Favus.
III. Alopecia.

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[1] Trans. Derm. Soc. Gt. Brit. and Ireland, vol. x., 1903-4, p. 34.

IV. Pediculi Capitis.
V. Seborrhœa.
VI. Canities.
VII. Hirsuties.
VIII. Tricoptylosis.
IX. Trichorrexis Nodosa.
X. Leptothrix.
XI. Tinea Imbricata.

I. **Ringworm** (syn. tricophytosis capitis, tinea tonsurans) starts as an insignificant, semi-bald, pink patch, usually overlooked, and when first seen by the physician is a white, powdery, circular spot or patch on the scalp of children, with *broken hairs*. The spot varies from the size of a threepenny piece to one denuding half the scalp. *Kerion* is a name given to a boggy suppurating patch on the head caused by ringworm.

Diagnosis. The broken hair stumps are quite characteristic. Dabbing chloroform over the part will reveal the diseased hairs which then look whitened like hoar frost. The diagnosis should be clinched by placing the hair on a slide, with a drop of liquor potassæ and examining under the microscope. The varieties of the fungus, however, can only be distinguished after staining by Gram's method (§ 623). A bald form of ringworm occasionally occurs resembling *alopecia areata*, but some broken stumps of hairs can be found at the margins of the patches. *Seborrhœa* has greasier scales and no ringworm fungus.

Etiology. The disease is rare in children over 14, and commonest under 10. Fair-haired children are more susceptible. Adults are practically immune. Animals, especially domestic animals, may contract ringworm and give it to children. The disease spreads rapidly in families and schools, and is due to a specific fungus. Two varieties of the fungus cause the disease—small spore (microsporon Audouïni) and large spore (megalosporon). See Figures 141 and 142, p. 876.

Prognosis. The disease lasts an indefinite time, but tends to disappear spontaneously about puberty. Much depends upon the stage at which the disease is first seen, the diligence of the treatment, and the variety of spore present—the small spore being much more intractable. Fair-haired children are more difficult to cure. The average duration is 1 or 2 years.

Treatment. Ringworm is a most difficult malady to cure, and great perseverance is required. The head should be shaved every

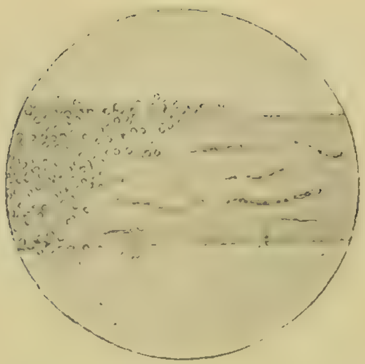

Fig. 141.—Small spored ringworm (Microsporon Audouïni) under a $\frac{1}{5}$th inch objective. Spores forming a thick-set sheath outside hair; mycelium within the hair branching and irregular, not parallel.

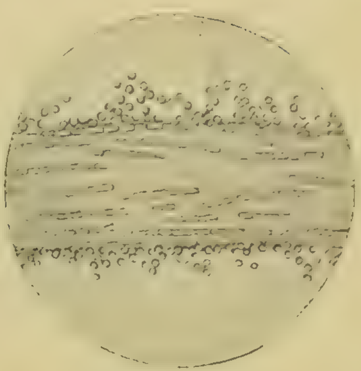

Fig. 142.—Large spored ringworm (Megalosporon). Mycelium within the hair parallel and breaking into equal segments with truncated ends.

Clinical Distinctions of the Two Varieties of Ringworm Fungus.

Small spore fungus (microsporon Audouïni).	Large spored fungus (megalosporon).
1. Hairs break off some way from follicular orifice, because spores eat away hair and grow downwards.	Hair breaks off quite short at or below follicular orifice, because fungus attacks hair root first and grows upwards.
2. Almost confined to scalp, but may invade body. The cause of 90 per cent. of the cases of ringworm of the scalp in England.	Chief cause of ringworm of body (tinea circinata), beard (tinea sycosis), and nails (onycho-mycosis).
3. Very intractable and highly infectious.	More readily cured, less infectious.
4. Never causes suppurative lesions (Sabouraud). English observers say it does occasionally.	Frequently pyogenic (*e.g.*, Kerion).
5. Only occasionally derived from horse, dog, cat.	Frequently derived from horse, dog, cat, etc.
6. Round spores, mosaic or irregular distribution, especially outside hair; mycelium branching and irregular within the hair.	Mycelium parallel, dichotomous, breaking up into equal segments with truncated ends within the hair.

ten days, and a well-greased linen cap worn which can be renewed every two or three days, the old one being burned. Some advise epilation. Patches seen very early may be aborted with strong parasiticides, *e.g.*, pure carbolic acid or iodine. Every day (1) cleanse and remove all debris with A.C.E. mixture; (2) apply parasiticides. Air and water should as far as possible be excluded. Amongst the numerous remedies may be mentioned salicylic acid, gr. x. to the oz. of collodion, and 10 per cent. hydronaphthol plaster applied in strips and covered with hydronaphthol jelly[1]—both these methods exclude air. Chrysarobin, carbolic, mercury, oil of cade and salicylic ointments are used in varying strengths, and it is well to change the remedies at times. Coster's paste once a month is useful.

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[1] Dr Morgan Dockrell, *The Lancet*, 1889, ii., 1110.

It is often very difficult to decide when a child is free from infection. My own method is to leave the case which appears cured without treatment and untouched for 10 days. If at the end of that time the surface is free from scaliness, the hairs are growing normally, and the microscope gives negative results, I recommend further treatment for a week, and then, after a second interval of 10 days, if the same tests answer, I pronounce the case as probably cured.

II. **Favus** occurs in the head and the body. It is very rare in England, but commoner in Scotland. It forms such characteristic irregular yellow crusts, with yellow, cup-shaped tops, and is accompanied by such a mousy smell, that the diagnosis is not difficult, even apart from the microscope, which reveals the spores and the mycelium of the achorion Schönleinii (Fig. 143). It develops slowly, is accompanied by itching, and leaves atrophic scars, while the hair becomes lustreless, brittle and falls out. It is less contagious than ringworm, but more intractable. The treatment is the same as in that disease, but epilation is more necessary.

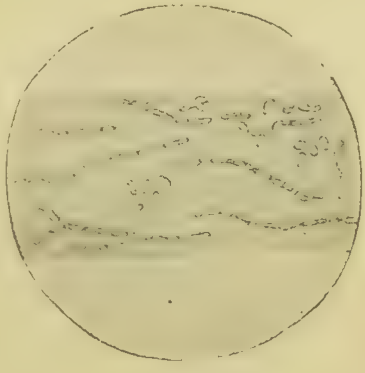

Fig. 143.—Favus fungus (tinea favosa or achorion Schönleinii) magnified about 300. Groups of irregular-sized spores within the hair; mycelium irregular in thickness and arrangement.

III. **Alopecia** (baldness) may be congenital or acquired, partial or complete, diffuse or in patches. The acquired condition may arise from (1) *premature senility*, in which the follicles are atrophied, and which usually begins in the vertex; (2) *general malnutrition*, as in tuberculosis, acute fevers, anaemia and nervous exhaustion; (3) *syphilis*, in which the baldness is disseminated or patchy in the early stages, or localised in the later stages of the disease (being then due to syphilitic lesions of the skin; (4) *favus and ringworm* (tinea decalvans), which affect the hairs and lead usually only to temporary baldness; (5) *lupus erythematosus*, in which the bald patches are permanent; (6) *psoriasis*, eczema, X-rays, etc., with only temporary loss of hair; and (7) *seborrhœa capitis*, which is probably the commonest cause of all.

Alopecia Areata is a special form of baldness occurring in circular patches which are smooth and white. Each patch slowly increases peripherally, and at the margin short diseased hairs may be seen, which have so characteristic an appearance as to enable us at once to identify the disease. The free end is of a normal thickness, but presents a ragged fracture where the hair has been broken off; from this point the shaft gradually becomes thinner towards the root, which is extremely atrophied. Thus it somewhat resembles a note of exclamation (!). Any part of the body may be affected. The disease runs a very protracted course, lasting if untreated for years. In course of time a few downy hairs begin to grow, white at first, but gradually becoming coloured. It is undecided whether the disease is of parasitic or neurotic origin.

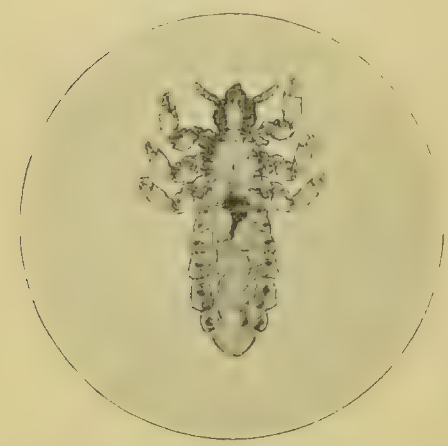

Fig. 144.—PEDICULUS CAPITIS × 10. It differs from the pediculus corporis only in being shorter and in its thorax and abdomen being more nearly equal in size (see p. 834).

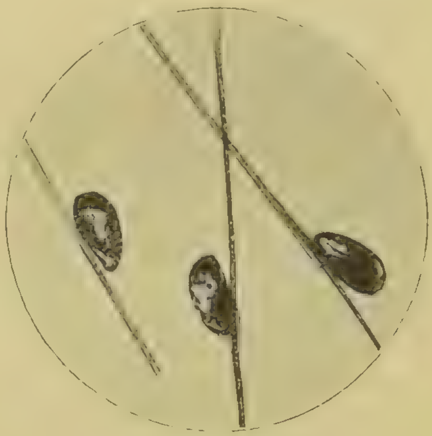

Fig. 145.—NITS (eggs) of PEDICULUS CAPITIS on hairs, slightly magnified.

In the *treatment* of baldness attend to the general health, and apply local remedies in order to stimulate the vascularity of the part by means of lotions or ointments, commencing with ammonia, turpentine and cantharides. If seborrhœa is present it must be corrected (see below). The treatment of the bald scars left by lupus erythematosus and other scarring eruptions of the scalp are hopeless, for the hair bulbs are as a rule destroyed. In alopecia areata the patient should be assured that nothing but a prolonged course of treatment is necessary. Faradisation and massage are useful.

IV. **Pediculosis Capitis** presents the following features—(1) the pediculi (see Fig. 144); (2) white specks on the hairs (the eggs or "nits"), which cannot be pulled off, by which they are distinguished from dandruff (Fig. 145); and (3) irritation and inflammation. If the condition is untreated there results pustulation with formation of thick crusts, matting of the hair, and enlargement of the occipital glands.

Treatment. The first indications are to kill the living parasite and also the nits or eggs. Both may be done by soaking the hair with methylated spirit or kerosene for 1 or 2 nights and combing the hair thoroughly with a small-toothed comb. Ungt. hyd. amm. (B. P.) or hyd. ox. rub. may also be used. The nits may be dissolved by washing the hair with strong vinegar.

V. **Seborrhœa Capitis** or disordered secretion of the sebaceous follicles may occur either in the form of "*seborrhœa oleosa*" (attended by excess of fatty secretion) or "s. sicca" (attended by diminished or inspissated secretion). S. capitis may or may not form part of a general seborrhœic dermatitis. It appears as dandruff, in localised patches or generalised over the scalp. It is diagnosed from eczema by the greater inflammation and exudation in the latter; and from psoriasis by the greater definition of the patches and the larger number and size of scales in psoriasis. The disease results in diffuse alopecia.

The *treatment* consists in washing the head once or twice a week with equal parts of soft soap and spirits of wine, and in rubbing in every night a lotion or ointment containing mercury, tar or sulphur.

VI. **Canities** or whiteness of the hair is, as the name implies, most usually an evidence of advancing years, or of overwork, sudden or prolonged grief, or neuralgia. The *treatment* is unsatisfactory apart from the improvement of the general health by tonics. The head should be examined for seborrhœa, or any other local disease, which, though it does not cause, may expedite the loss of colour.

VII. **Hypertrichosis** (syn. Hirsuties) is a growth of hair either abnormal in amount or in position, and the most troublesome of these is the moustache or beard found on the faces of some women. *Treatment* consists of the removal either by the razor or electrolysis, or in the use of some paste, consisting of sulphate of barium or calcium.

VIII. **Trichoptylosis**, or splitting of the ends of the hairs, is met with sometimes in women, and is probably caused by an undue dryness of the scalp, though it often occurs in association with a deranged general health.

IX. **Trichorrexis Nodosa** is a very rare disease in which a series of spindle-shaped swellings appear upon the hair, which break transversely, and leave a brush-like extremity. It is due to a microbe.

X. **Leptothrix** (syn. Mycosis axillaris, trichomycosis nodosa) is a disease affecting the hairs of the axillæ and scrotum. The hairs are dry and knotty, due to adherent small concretions, which may affect the whole length of the hair, but not the follicle, or may occur as separate nodules on a hair. Bacilli are found in these concretions, and the hair may be split longitudinally (Fig. 146).

XI. **Tinea Imbricata** is a contagious disorder of the tropics due to a fungus affecting the axillæ for the most part. There is an eruption consisting of rings within rings.

§ 498. General Remarks on the Treatment of Skin Diseases. There are 3 principles upon which we must depend for success: (1) If we except mercury and iodides in syphilis, and perhaps arsenic in lichen planus, pemphigus, and dermatitis herpetiformis, there are no such things as specific remedies in skin diseases. It is therefore not sufficient for purposes of treatment to diagnose a case as eczema, psoriasis, lupus, etc. We must recognise *the stage of the disease* and the *precise pathological process* before us. An ointment which would cure a chronic eczema would greatly aggravate an acute weeping one. It follows, therefore, that it depends not so much upon the name which we decide to give to an eruption, as upon the amount of congestion, swelling, scaling, thickening, discharge, itching, etc., which is present. We have, in a word, to treat the symptoms, the sum of which constitutes the disease at that particular moment. This requires very considerable experience and herein lies one of the justifications of the speciality of dermatology. (2) The *method* of application of a

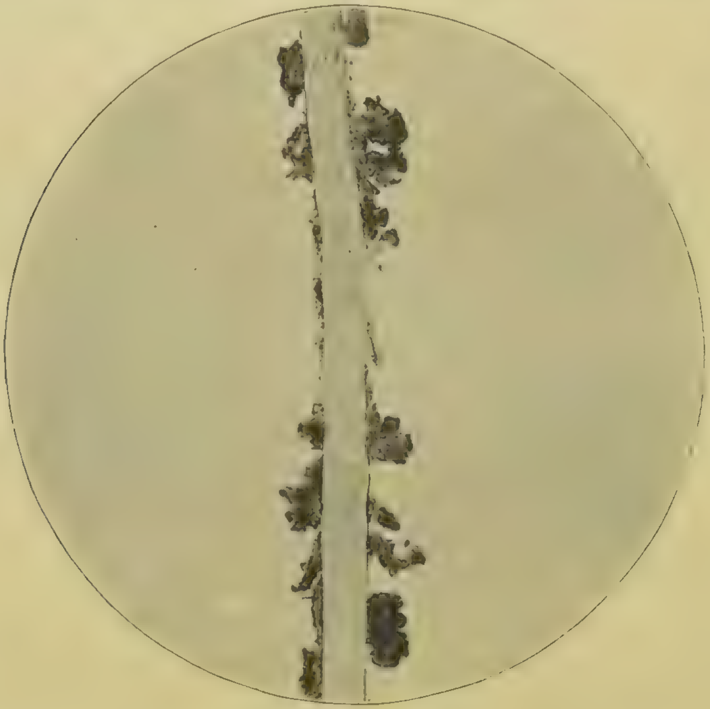

Fig. 146.—Leptothrix, slightly magnified.

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remedy is of quite as much importance as the composition of it. (3) The *idiosyncrasy* of a patient and the susceptibility of his skin to various remedies must always be remembered. This is especially true of the face, for what will benefit one person's skin will irritate another's.

Bearing in mind these three important considerations, it may be convenient to divide external remedies into 3 groups, the first being chiefly indicated for acute, congestive or moist conditions, the second for dry, scaly, hard and chronic conditions, and the third group (caustics) having a corrosive action for the removal of diseased structures.

(a) SEDATIVES and ASTRINGENTS are used chiefly in acute conditions to reduce hyperæmia, to check exudation, and to allay pain. In order of increasing strength the principal ones are zinc, lead, bismuth, mercurous, and, in a less degree, mercuric and silver salts. Zinc and bismuth are inert on the unbroken skin. These remedies are generally applied in the form of ointments, as a powder, or still better, suspended *in lotions* for acute eczema and similar moist conditions. Zinc oleate is also a bland preparation, and zinc gelatine may be used when there is little discharge. Ung. diachyli is also soothing. Mercurial preparations are more efficacious in chronic conditions. The perchloride is irritating and if used strong comes under the heading of caustics. Vegetable astringents are not much used, though the glycerine of tannic acid and boracic acid may be used for congestive conditions around the mucous orifices.

(b) STIMULATING APPLICATIONS, ANTISEPTICS, TARS, and AROMATICS are useful to stimulate chronic conditions to more healthy action. They include wood tar or creasote and coal tar or creolin, carbolic acid, resorcin, thymol, β-naphthol, benzoic and salicylic acids—salicylic acid especially being a very useful preparation to reduce hyperplasia of the epidermis—ichthyol, sulphur, and chrysarobin. The earlier named of these are less irritating than the later ones; indeed weak preparations of tar may act as a sedative.

(c) CAUSTICS have a definite corrosive action upon the skin. Thus liquor potassæ may be used to remove the superfluous epidermal scales of psoriasis prior to the application of an ointment or plaster. Others are mercuric chloride, acid nitrate of mercury, nitric acid, pure carbolic acid, etc. Soft soap is the mildest of such preparations and may be usefully added to an ointment for the treatment of inveterate psoriasis.

(d) PROTECTIVE MEASURES. The old-fashioned paste, *i.e.*, an ointment made up with a large proportion of some powder (*e.g.*, F. 75), comes under this heading. Pastes are applied by laying on in a thick layer. One of the most useful forms of protection is ZINC GELATINE. Painted on to the diseased skin this fulfils four indications—(i.) gentle compression and support, as in varicose eczema or hypostatic congestion; (ii.) protection from the action of the air or friction while allowing natural evaporation and healthy action to go on beneath; (iii.) it allays itching; (iv.) is an excellent dressing and means of applying remedial agents constantly. Another protective agent is the PLASTER MULL introduced by Unna, which consists of reagents mixed with gutta-percha and some basis, and spread upon a piece of muslin.

CHAPTER XIX.

THE NERVOUS SYSTEM.

THE nervous system is the governing and controlling power of the body and permeates every other system and structure. As a consequence the investigation of its diseases necessarily requires a wide knowledge, and it also presents a certain amount of difficulty. Neurology does not admit of narrow specialism; a cerebral or spinal hæmorrhage, for instance, is in its etiology and treatment cardio-vascular, and for the complete investigation and treatment of diseases of the nervous system a thorough knowledge of general medicine is indispensable. The difficulty of investigation, however, is more apparent than real if the scheme of studying medicine adopted in this work be followed. Our first duty when dealing with a widespread structure like the nervous system is to *localise* the seat of mischief, and this we are enabled to do in a large proportion of instances by a moderate acquaintance with the anatomy and physiology of the nervous system. This accomplished, the 2nd stage of the process, the diagnosis of the *nature of the lesion* is not generally difficult, for the position it occupies, the age of the patient, his history, and the mode of onset of the illness will generally afford us fairly certain bases for decision. A convenient method of examining a case of nervous disease will be given below, and by bearing in mind the two steps just referred to the student should not experience much difficulty in diagnosis.

Anatomical and Pathological Preliminaries.

§ 499. **The Neuron.** The central nervous system consists of brain, spinal cord, and nerves. But such a division is purely artificial, and the best way is to regard the nervous system as consisting of a collection of cells with long processes (Waldeyer). A nerve cell with its various processes is called a *neuron*, the cell being called the *neuron-body* and the processes named *dendrons* (Fig. 147). One of these dendrons is longer and larger than the others, and receives a special covering in the shape of a medullary sheath after leaving the neuron-body or cell; this process is called the axis-cylinder process or *neuraxon.* The neuron-bodies or cells are found entirely in the *grey matter* of the brain and cord, or in the various ganglia such as those of the posterior roots. The neuraxons in passing to their destinations are often of considerable length; they pass through and constitute the *white matter* of the brain and cord, where they form the nerve fibres, and finally traverse the *nerve trunks* to their destination in the muscles, skin, or special senses. The words "nerve fibre," "neuraxon," and "axis-cylinder process" are synonymous; and the words "ganglion cell," "multipolar cell," "neuron cell," and "neuron-body" are also synonymous.

Connections of the Neurons. A neuron such as that just described constitutes a unit of the nervous system, and immense quantities of such units go to form nerve matter, wherever it may be found. The different cells at one time were thought to be connected with each other by means of the branching ends (dendrites) of their processes. Doubt has recently been thrown on this, but whether there is organic connection between the different neurons or not their dendrites interlace and lie side by side, and a nerve impulse can pass from one to another, either directly or after the manner of an electrical inductive process.

Nutrition of the Neuron. A nerve cell depends for its nutrition on the quality of the surrounding matrix,

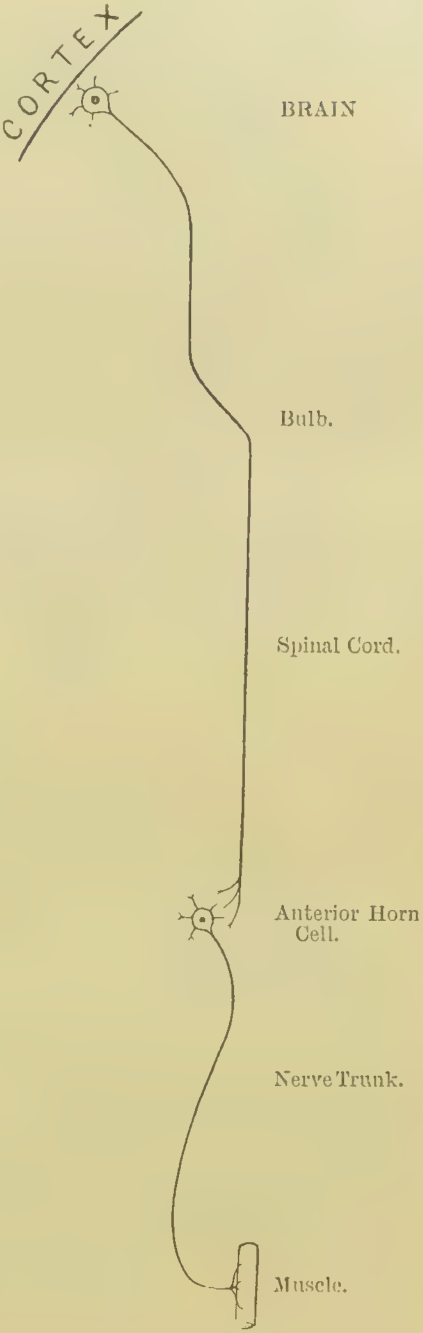
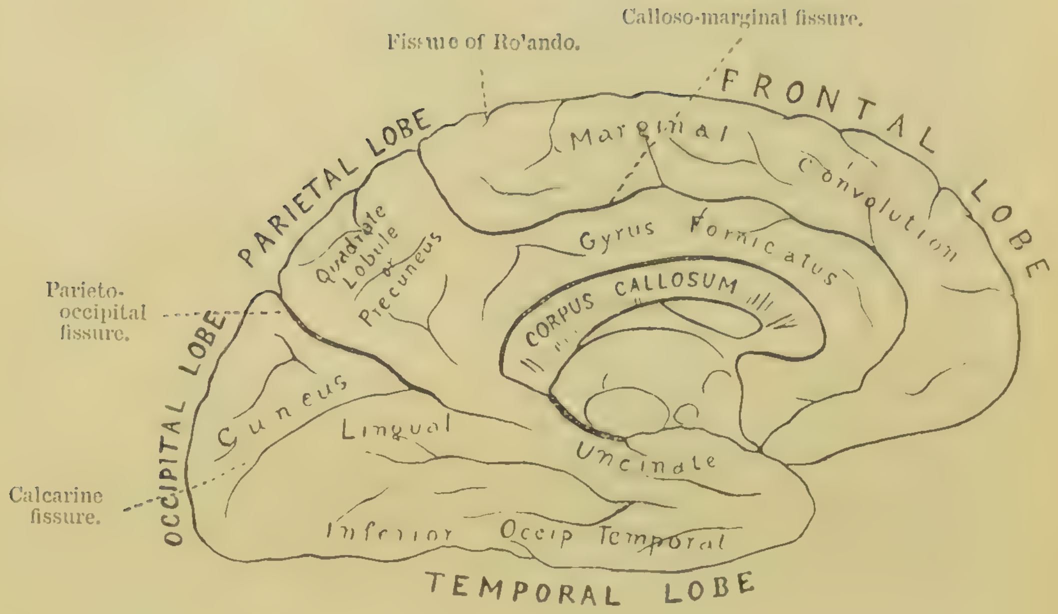
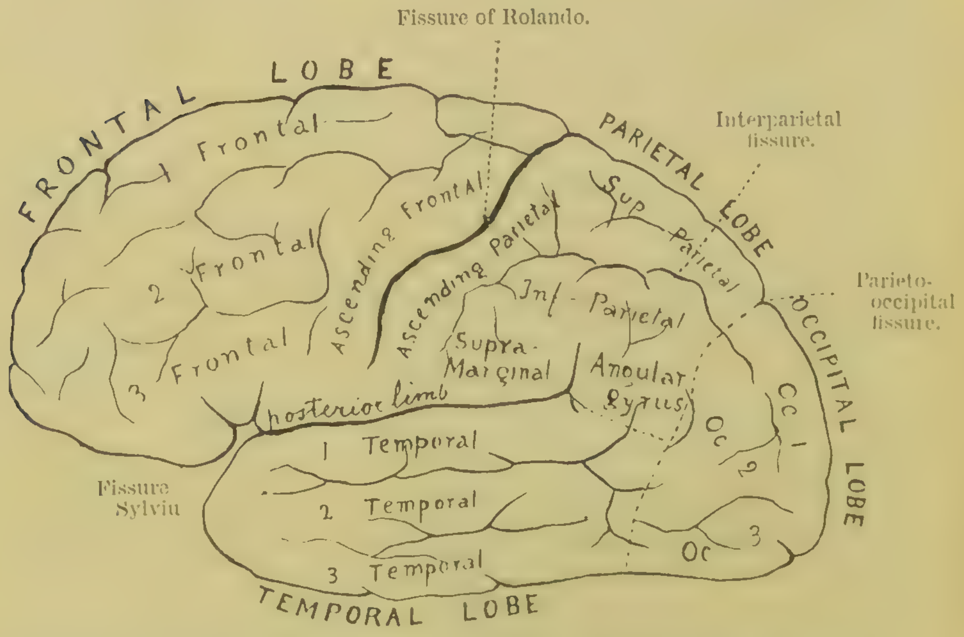


Fig. 147.—Diagrammatic representation of a MOTOR NEURON, the upper level (UPPER MOTOR NEURON) being from brain to spinal cord, the lower level (LOWER MOTOR NEURON) being from anterior horn to muscle. Compare Figs. 152 and 162, *i.e.*, motor tract and path of tabes.





Figs. 148 and 149.—Convolutions and fissures of the brain, external surface (lower figure) and median aspect (upper figure) of the left cerebral hemisphere. The uncinate lobule is sometimes called the gyrus hippocampi. This with the gyrus fornicatus together form the falciform lobule. The paracentral lobule is the posterior part of the marginal convolution.

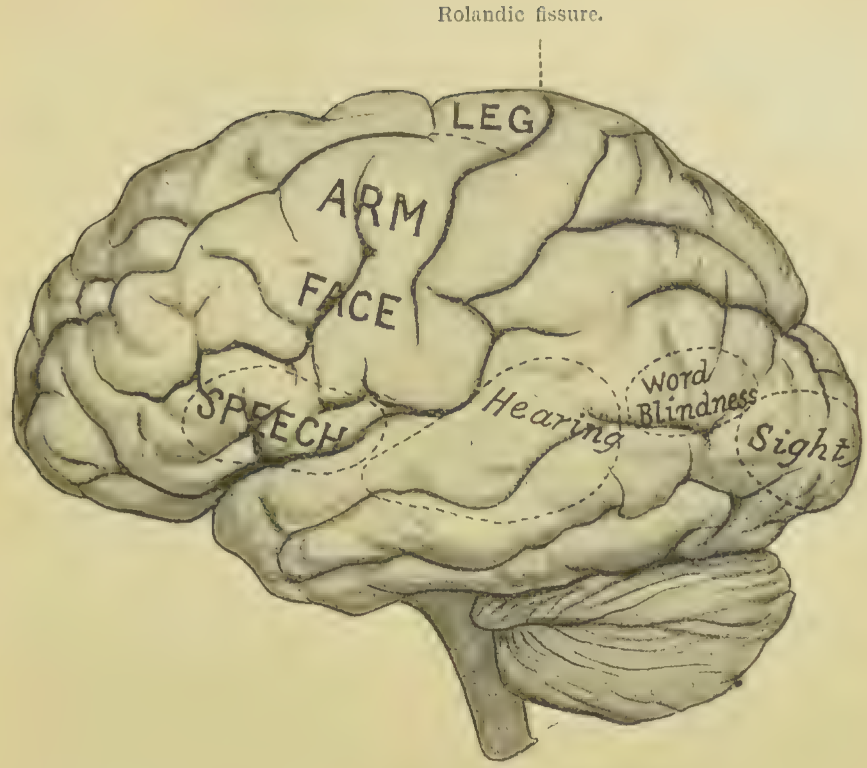


FIG. 150.

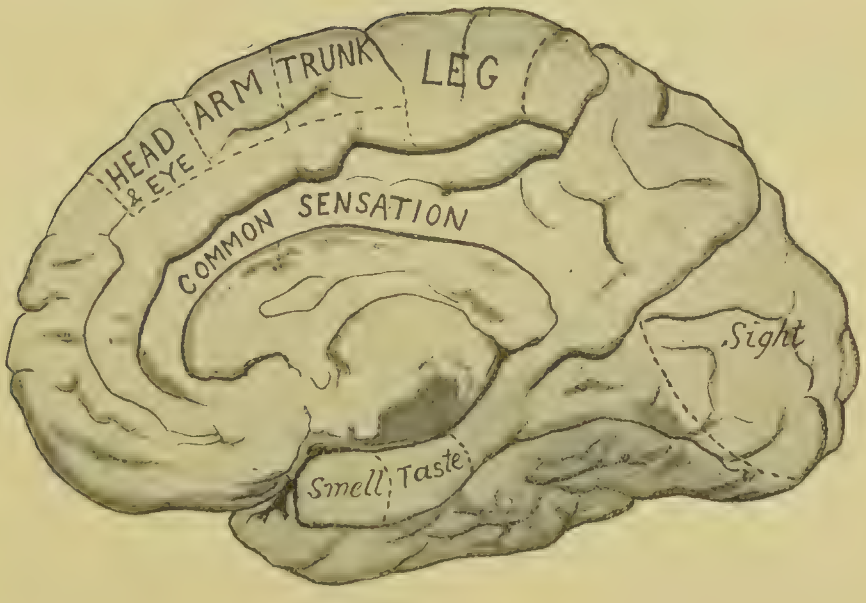


FIG. 151.

Figs. 150 and 151.—LOCALISATION OF THE CHIEF FUNCTIONS on the cerebral cortex. The motor area was formerly located both in front and behind the fissure of Rolando, but Sherrington and Grünbaum's researches (Trans. Path. Soc. Lond., 1902), confirmed by Alfred Campbell and others, have shown that it lies wholly in front of this fissure. The upper figure is the external and the lower is the median aspect of the right hemisphere.

just as the nutrition of a tree is modified by the soil surrounding its roots. When a nerve fibre is divided the part on the side farthest from the cell of origin rapidly degenerates (Wallerian degeneration). It is evident therefore that the cell body has a powerful control over the nutrition of the nerve fibre to which it gives origin. This nutritional dependence of the nerve fibres on their cells of origin is one of the leading facts in neuropathology, and it accounts for those *descending* and *ascending* degenerations which are met with so frequently (Figs. 152 and 153). Moreover, by means of the artificial division of nerve tracts histologists have been enabled to trace the course of nerve fibres in the brain and cord which would be impossible of dissection.

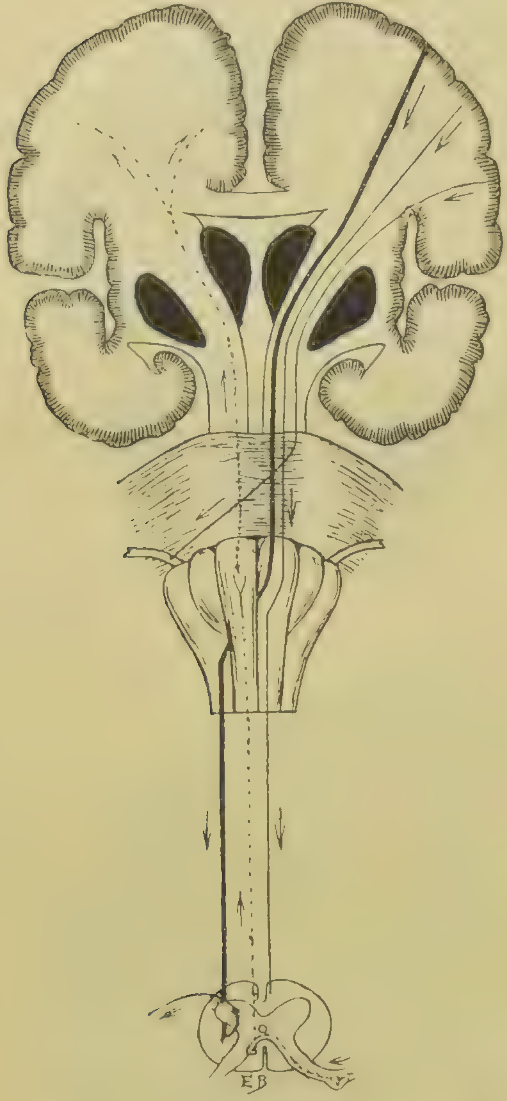


Fig. 152.—Motor (descending arrows) and sensory (ascending arrows) tracts. The motor tract showing the course of the facial fibres, the crossed pyramidal tract (thick), and the direct pyramidal tract. The sensory tract shows the crossing, at the spinal level, of the sensations of touch and pain.

Waldeyer's neuron scheme of the nervous system as above depicted is not regarded by everyone as the most scientific, but for clinical purposes it is the most lucid and practical existing at the present time.

§ **500.** The **Motor Tract.** Looked at in its simplest form, the **encephalon** consists of a mass of white fibres (the corona radiata) spreading out towards the surface like a fan, or the petal of a tiger lily, the edges of which are covered by the grey cerebral cortex. This surface or cortex is amplified by means of foldings (convolutions) which, in the genus homo are more numerous than in any other of the vertebrata (Figs. 148 and 149). Unlike the spinal cord, the grey matter of the brain is found chiefly upon its surface. But in the interior, at its lower part, there are three groups of grey matter which, from before backwards, are the corpus striatum, the optic thalamus, and the corpora quadrigemina (two on each side). The corpus striatum is divided into two grey masses (the caudate nucleus internally and the lenticular nucleus externally) by a most important band of white matter, the *internal capsule*, which carries the conducting strands from one side of the brain to the opposite side of the body and limbs (Fig. 152). Internal to the lenticular nucleus comes the anterior end of the thalamus.

In regard to its *functions* (Fig. 150) the cortex of the brain may be divided roughly into three parts, anterior, posterior, and middle third. The anterior third, or frontal portion, is the seat of the intellect. Gross lesions in this position may exist for a long time without any symptoms other than dulness and stupidity, headache, and perhaps vertigo and nausea.[1] The posterior third, or roughly the occipital portion, is the seat of vision and several other important sense perceptions. The middle portion just in front of the fissure of Rolando forms the motor area. The different centres for movements of the limbs on the opposite side are shown in Figs. 150 and 151. It will be observed (and it will enable us to remember the position of these centres) that the most complex movements, those of the lips and tongue, are farthest forward and lowest down, nearest the frontal or intellectual region. Behind and above this, in order, come the centres for the face, arm, leg, and (on the median aspect of the hemisphere) the trunk. This order, it will be observed, is that of less and less complexity of movement.[2] These centres are not absolutely defined, but overlap each other. This region is called the motor area because—(1) electrical stimulation in animals gives rise to movements of the limbs; (2) irritative lesions give rise to convulsions; and (3) destructive lesions in this position cause paralysis of the respective limbs.

DISEASES OF THE BRAIN may be classed under circulatory lesions, tumours, inflammatory lesions, and functional diseases. Vascular lesions (hæmorrhage, embolism, thrombosis) are of sudden, tumours of more gradual, onset. Functional diseases include hysteria, epilepsy, insanity, and neurasthenia. Syphilis may affect the brain in four ways—endarteritis, leading to thrombosis, meningeal affections, gummata, and the mental symptoms of paralytic dementia. Gummata constitute one of the most frequent forms of cerebral tumours. See also § 503.

Medulla and **Spinal Cord.** Those parts of the bulb and the pons varolii which form the floor of the fourth ventricle and the iter a tertio ad quartum ventriculum contain a series of grey nuclei which give origin to the cranial nerves. These form, as it were, a continuation upwards of the anterior and posterior horns of the grey matter of the spinal cord. Indeed, if we imagine the spinal cord to be split from behind, and, as it is traced upwards, to be opened outwards, so that the posterior grey cornua come to be external, and the anterior columns come to the surface beside the middle line, we should find, what is actually the case, that the motor nuclei of the 12th, 11th, 7th, 6th, and the motor nucleus of the 5th cranial nerves (corresponding to the motor anterior horns) lie on each side of the middle line; and that the sensory nuclei of the 10th, 9th, 8th, and 5th nerves (corresponding to the posterior horns) lie in a more external situation. The nuclei of the other motor nerves (4th and 3rd) lie much further forward beneath the aqueduct of Sylvius.

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[1] Apathy and somnolence were absolutely the only symptoms in a case shown by Dr. Ferrier at the Neurological Society in 1892; and in two cases under my own care at the Paddington Infirmary, in 1892, verified by autopsy. These symptoms occur in monkeys deprived of their frontal lobes, but they sometimes recover completely, and regain their faculty of attention.

[2] There is a possible exception to this general statement in that electrical stimulation of the first and second frontal convolutions gives rise to conjugate deviation of the head and eyes, and that certain parts of the inner aspect of the brain give rise to movements of the arm.

Functions. The bulb contains important reflex and automatic centres. The *reflex centres* are for the closure of the eyelids, sneezing, coughing, sucking, mastication, secretion of saliva, swallowing, vomiting, dilatation of the pupil, and the vaso-motor regulation (general dominant reflex centre). The *automatic* centres are respiratory, cardio-inhibitory, vaso-motor, sweating, and convulsion centres. The convulsion centre is situated just where the medulla joins the pons; stimulation of it causes general spasms. The centre may be excited by a venous condition of the blood, as in asphyxia, anæmia (as when the carotids are tied), or congestion, as in compression of the veins coming from the head.

The **spinal cord** in the adult extends from the margin of the foramen magnum to a point opposite the first lumbar spine, and measures 16 inches. In the infant it extends to the bottom of the spinal canal. There are enlargements in the cervical and lumbar regions, containing the motor cells for the muscle supply of the limbs. In the *white* matter of the cord, which is concerned in conducting impulses upwards and downwards, certain columns have been mapped out by means of developmental and experimental inquiries, and by studying the position of secondary descending and ascending degenerations—these are shown in Fig. 153. The best way to regard the *grey* matter of the cord is as a series of segments or discs, superimposed one upon the other, 32 in number, corresponding to the pairs of the spinal nerve roots, each segment being concerned in the three functions of the cord, viz., conduction, reflex action, and the regulation of certain neuro-muscular phenomena.

MOTOR IMPULSES (Fig. 152). It is important to remember that a motor impulse passing from the cerebral cortex to the periphery must pass through two neurons—(1) the *upper motor neuron*, connecting the cortex with a multipolar cell in the medulla or cord; (2) the *lower motor neuron*, which consists of the multipolar cell in the anterior horns of the cord, and the nerve fibre from this to the muscle fibres. A motor impulse starting in the Rolandic area passes through the corona radiata in the central white matter of the brain, through the internal capsule—in which situation all the motor fibres are collected into one small bundle occupying the anterior two-thirds of the hinder limb—through the middle two-fifths of the crus cerebri of the same side, through the pons in a band lying between the superficial and deep transverse fibres, and through the anterior pyramids of the medulla. Here the motor fibres cross to the opposite side (at least the bulk of them do), to form the *crossed pyramidal tract* in the lateral columns of the spinal cord, which diminishes in size from above downwards as the terminal dendrites come into relation with those of the anterior multipolar cells (the dark band in Fig. 152, and Fig. 153). This terminates the upper motor neuron (cerebro-spinal neuron) of the muscles of the trunk and limbs. At the point of decussation in the medulla a few of the motor neuraxons, instead of crossing over, pass down the same side of the cord in the anterior column close beside the anterior fissure, forming the *direct pyramidal tract* which terminates about the middle of the dorsal region. The lower motor neurons (the spino-muscular level) start in the multipolar cells of the anterior horns (or their analogues, the motor cranial nuclei) and pass out through the anterior nerve roots (or the cranial nerves) into the peripheral nerve trunks, and terminate in the muscles. The total number of fibres (neuraxons) of the lower level passing out through the anterior roots is far greater than the number of neuraxons in the upper level; it follows therefore that one neuraxon of the cerebro-spinal series must control several associated neurons of the spino-muscular level.

Taking a *horizontal section of the internal capsule*, the motor fibres in the genu supply from before backwards the eyes, face, and tongue. The motor fibres in the anterior two-thirds of the posterior limb of the capsule supply from before backwards the shoulder, arm, hand, trunk, and leg.

DESCENDING SCLEROSIS. The cortical cells preside over the nutrition of the neuraxons as far down as the anterior horns, and consequently a disease of the cortical cells, or a lesion cutting off these cells from the fibres below, will be attended by a degeneration (**descending sclerosis**) down the lateral column to the ends of these neuraxons. The cells in the anterior horns of the spinal cord similarly preside over the nutrition of the peripheral nerve fibres; and disease of these cells, or a lesion cutting them off from the neuraxon below is followed by degeneration of the motor nerves and atrophy of the muscles with which they are connected.

Three important clinical facts follow from the foregoing which the student must always remember. First, that a lesion in the small motor band which we call the **internal capsule** (a very common position for vascular lesions) will produce extensive paralysis of the limbs, face, and tongue of the opposite side (hemiplegia).

Secondly, all paralyses due to lesions of the **upper motor neurons**, situated anywhere between the cortex and the anterior horns, are attended by muscular rigidity, with increase of reflexes, but without muscular wasting, because the multipolar cell of the lower neuron remains intact. Consequently, all hemiplegic lesions and paraplegic lesions attended by lateral sclerosis are recognised by being RIGID, NON-ATROPHIC, with INCREASED DEEP REFLEXES.

Thirdly, the anterior multipolar cells which start the lower neuron, preside over the nutrition not only of the peripheral nerves, but also of the muscles with which they are connected, and consequently **lower motor neuron** lesions on the other hand are characterised by being FLACCID, ATROPHIC, with LOSS OF DEEP REFLEXES.

System Lesions of the Cord. From the above it follows that diseases attacking certain tracts or columns of the cord are accompanied by symptoms indicating destruction of the function performed by those tracts. Such diseases are named System Diseases, and differ from gross lesions (such as transverse myelitis) which involve a *horizontal segment* of the cord, in that they select *vertical* tracts or columns of the cord.

TABLE OF SYSTEM LESIONS OF THE CORD (Fig. 153).

Symptoms.	*Tracts affected.*	*Types of Disease.*
Rigidity, increased reflexes, no wasting.	Crossed pyramidal tract.	Lateral sclerosis.
Ataxy, loss of reflexes.	Posterior columns.	Tabes dorsalis.
Flaccidity, loss of reflexes, wasting, and R. D.	Anterior horns.	Anterior poliomyelitis, acute and chronic.
	Combined System Diseases.	
Ataxy, rigidity, increased reflexes, no wasting.	Posterior columns, direct cerebellar and crossed pyramidal tracts.	Ataxic paraplegia.
Rigidity, increased reflexes, some wasting.	Anterior horns and crossed pyramidal tract.	Amyotrophic lateral sclerosis.
Ataxy, tremor and loss of knee jerk.	A combined disease of posterior and lateral tracts.	Friedreich's hereditary ataxy.

§ **501. Sensation** and the **Sensory Tract.** The sensory or centripetal tract is not so clearly known as the motor, partly because of the difficulty of accurately testing it in man and animals, partly because *loss of sensation in man is more rapidly restored than loss of motion.* The leading facts to remember about common sensation (tactile, painful, and thermal) are that it enters the cord by the posterior roots (Fig. 152, *ante*), and crosses to the opposite side soon after entering, so that a unilateral lesion of the cord produces paralysis of the same, anæsthesia of the opposite side (Brown-Séquard's paralysis). The exact path by which sensations of touch, pain and temperature reach the brain is not yet certain. The anatomy of the muscle-sense neuron will be referred to below, § 502, and under tabes dorsalis (§ 572). There are at least three levels of sensory neurons, but lesions at their different levels are not distinguishable as in the case of the motor neurons.

After entering the cord from the periphery the sensory neuraxons take several different routes. (1) One set of fibres enters the postero-external (Burdach's) column and divides into branches which ascend and descend a short distance, then pass either into the grey matter of the cord or into the postero-median (Goll's) column and go up on the same side to the medulla by Goll's column. (2) Other fibres end in the grey matter of the cord round cells whose axis-cylinders cross and ascend the cord in the antero-lateral (Gowers') tract. (3) The direct cerebellar tract. (4) Another set of fibres passes into the grey matter of the cord and ends round the anterior horn cells, thus forming the path of the reflex arc. *The first three tracts pass up to the medulla.* The direct cerebellar tract then passes on to the cerebellum. Goll's and Burdach's columns form the funiculi in the medulla, and fibres from these cross in the medulla and pass up together with Gowers' column and the 5th cranial nerve (which joins the sensory tract at that part) in the lemniscus or fillet which occupies *the posterior part of the pons*, through the *tegmentum of the crus* cerebri, to the *basal ganglia*, where some of the fibres end. The sensory tract in the brain then passes up through the posterior third of the hinder limb of the *internal capsule* (sensory crossway), coming, be it observed, from the opposite limbs. In this position it receives the visual and auditory fibres from the optic and auditory nerves, and probably also from the nerves of smell and taste, all from the opposite side. The sensory tract continues upwards through the corona radiata, and some say that the sensory tract from the body ends in the Rolandic or motor area. But Horsley has experimentally located common sensation in the gyrus fornicatus (Fig. 150), and clinical experience tends to show that the centres for touch at least are situated in the gyri fornicati and hippocampi.[1]

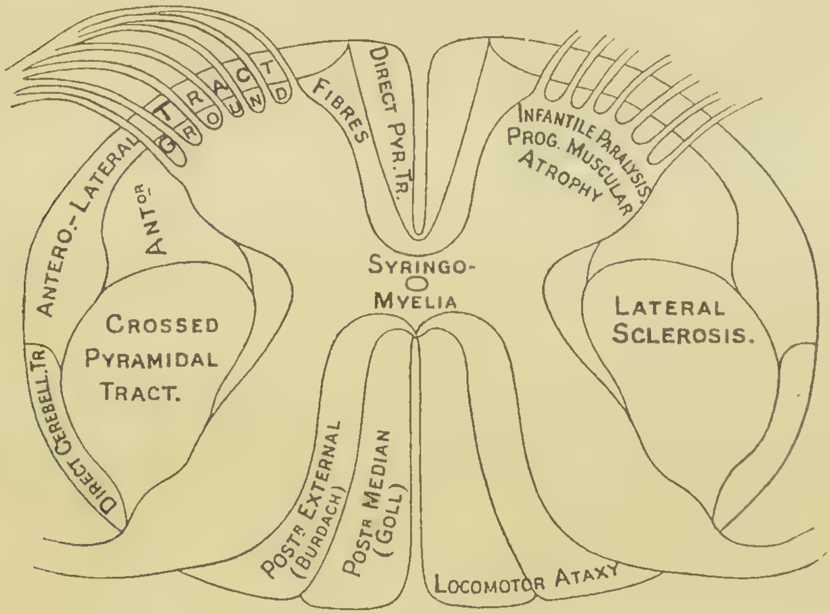

Fig. 153.—Transverse section of spinal cord in the cervico-dorsal region. Descending degenerations take place chiefly in the crossed pyramidal tract, direct pyramidal tract: chief ascending degenerations in the columns of Goll and Burdach, Gowers' tract, direct cerebellar tract.

Ascending Degeneration (Ascending Sclerosis) is found in the columns of Goll and Burdach, the antero-lateral tracts, the direct cerebellar tracts, and sometimes in the comma-shaped tract, secondary to lesions in the cord lower down.

§ 502. The **Cerebellum, Co-ordination** and **Equilibrium.** In order that our balance in the erect posture may be preserved and our different muscular movements effective, the contractions of the various muscles must be co-ordinated or correlated to one another. The centre for this co-ordination appears to lie in the cerebellum, and especially in its middle lobe. The cerebellum consists of two hemispheres and a middle lobe. It is connected above with the cerebrum by the superior peduncles, below with the medulla by the inferior peduncles, and in front with the pons varolii by the middle peduncles—cerebro-afferent fibres. In order to co-ordinate the various movements and to preserve equilibrium of the body, the cerebellum must be kept correctly informed of the relation we bear to surrounding objects, and also as to the state of contraction of the various muscles. This is accomplished by four different sets of afferent or in-going impulses. (1) The *sensation* of touch of surrounding objects is transmitted through the paths of common sensation. (2) The *sight* of surrounding objects informs us of our relation to them, and therefore it is assumed centripetal fibres pass from the centres for movement of the eyeballs and sight to the middle lobe of the cerebellum, to assist in regulating the attitude of the body so far as it relates to the maintenance of our proper distance from the objects around us. It is possibly in this way that nystagmus comes to be a symptom of cerebellar tumour.

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[1] *E.g.*, two cases recorded by the author in *Brain*, part xiv., 1891, p. 270, and part xv., 1892, p. 448.

(3) *Orientation*, or the position in which our body lies at any given moment in relation to vertical and horizontal planes, is recognised by impulses coming from the semi-circular canals, transmitted through the vestibular portion of the auditory nerve to the cerebellum. Disease of the semi-circular canals or of this nerve produces a feeling of giddiness or unsteadiness, and there may be an inability to stand (*e.g.*, in Ménière's disease).

(4) *Muscle-Sense.* A knowledge of the state of contraction of our muscles is recognised as muscle-sense, and it is necessary for the preservation of equilibrium and co-ordination. This muscle-sense tract starts, we now know, in the muscle spindles.[1] It passes through the nerves into the spinal cord by the posterior root, up the columns of Goll (postero-median columns), and the direct cerebellar tracts to the middle lobe of the cerebellum. The columns of Goll transmit muscle-sense impressions from the lower extremities; the direct cerebellar tracts those from the upper dorsal and cervical regions chiefly. Interruption of these muscle-sense tracts is the principal factor in the production of tabes dorsalis.

Destruction of any one of these four tracts disturbs equilibrium more or less. In tabes dorsalis there is generally anæsthesia of the soles of the feet (1), and the muscle-sense (4) is also disturbed, so that when the eyes are shut and path 2 is interrupted, the patient tends naturally to fall (Romberg's sign).

Cerebello-Efferent Tracts. The cerebellum only regulates, it does not initiate, muscular contractions. These are started by the cerebrum, and are merely controlled by the subordinate centre in the cerebellum. The connection of the cerebellum with the cerebral hemispheres is mainly a crossed one, fibres passing from the cerebellar hemisphere of one side to the cerebral hemisphere of the opposite. *Lesions of the cerebellum* produce (1) a staggering or reeling gait, and a deficiency in equilibrium, but they do not produce absolute paralysis. Lesions in the upper part of the middle lobe produce a tendency to fall forwards; those of the lower part to fall backwards; and if of the lateral lobe, the patient may show a tendency to fall *towards* the affected side, or there may be forced rotary movements towards the affected side. Tumour of the lateral lobe may produce inco-ordination and paresis of the limbs on the same side, from pressure on the motor tract or from affection of the contra-lateral cerebral centre.

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[1] F. E. Batten, *Brain*, part xx., 1897, p. 138.

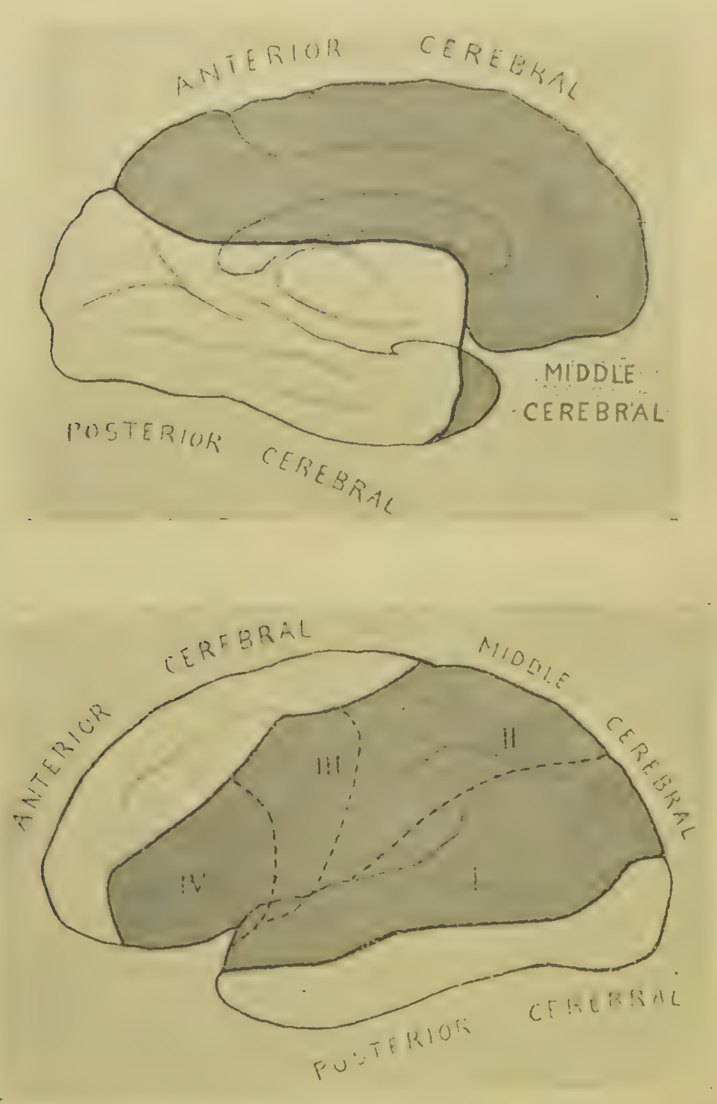
§ 503. **The Membranes of the Brain and Spinal Cord** should be mentioned together as they are identical in structure, continuous with one another, and subject to the same diseases.

The membranes of the brain and cord are subject to a great many *lesions*. (1) Meningitis is inflammation of the membranes; several different forms are recognised. (i.) External, or pachymeningitis begins and predominates in the dura. (ii.) Internal or lepto-meningitis is inflammation which begins and predominates in the pia and arachnoid. (iii.) A specific epidemic form of meningitis is recognised, which consists of a lepto-meningitis in which the pneumococcus of Fraenkel has been found. (iv.) A septicæmic form of meningitis is recognised which is apt to complicate scarlatina and other acute diseases. Meningitis has also been traced to syphilis and bacterial processes. (2) Tubercle is one of the commonest diseases affecting the meninges in children. It involves the pia mater and arachnoid, and almost invariably starts and predominates in the transverse fissure and fissure of Sylvius. (3) Syphilitic gumma of the meninges is the commonest meningeal disease in adults. Syphilis also produces simple thickening of the meninges and disease of its arteries. (4) Cancer chiefly affects the dura mater secondarily to cancer in other parts. (5) Fibrous thickening of the dura is found as a chronic form of pachymeningitis. (6) Bony plates may be found as the result of a chronic meningitis. I have generally found them in the arachnoid, and chiefly in old people. (7) Hæmorrhage may take place in both the meninges of the brain and cord. In the former position it is known as hæmorrhagic pachymeningitis, and in the latter hæmatorrhachis. The blood may be effused in three positions; it may be extradural, subdural, or subarachnoid. (8) Finally, the meninges of either brain or cord may be the seat of injury.

In all these disorders the disease may start or predominate either in the cranium or the spine, but it is very apt to spread to the other. It must also be remembered that the surface of the brain and spinal cord derive their nourishment almost entirely from the pia and arachnoid which invest them, and therefore diseases of the membranes impair their nutrition considerably. And since the cortex of the brain is the most important part functionally, grave consequences may ensue from meningeal diseases.

§ 504. **The Cerebral Circulation.** The ARTERIES of the brain are derived from two internal carotids and the vertebrals, which form the circle of Willis (Fig. in § 546). The middle cerebral is the most important artery of the brain, and it is a continuation onwards of the internal carotid. The internal carotid (which arises from the highest part of the aorta) and left middle cerebral are in a direct line for emboli from the valves of the heart. There are two series of branches of the middle cerebral, the external branches and the internal branches. The sylvian artery, which constitutes the chief supply of the internal capsule, is also in the direct line from the heart, and as it feels the full ventricular shock it is frequently the seat of hæmorrhage. For the same reason also it is frequently the seat of embolism in younger persons, and this accounts for the greater frequency of right hemiplegia in circulatory lesions. Figs. 154 and 155 show the vascular areas supplied by the cerebral arteries.

VEINS and SINUSES. The venules collect the blood into veins of the brain (which have no valves) which enter the cerebral sinuses backwards. Most of the blood leaves the cranial cavity through the internal jugular veins, which are continuations of the lateral sinuses. Should they be blocked, there are a few collateral communications through which the antracranial circulation communicates with the veins outside the skull. Thus, (1) the interior end of the superior longitudinal sinus communicates with the veins of the nose; (2) the ophthalmic veins communicate through the orbit with the facial veins; (3) the lateral sinus communicates with the occipital veins through the mastoid cells; (4) the superior longitudinal sinus communicates through the calvarium with the veins of the scalp; and (5) the inferior petrosal sinus communicates with the deep cervical veins. The veins of Galen collect the blood from the collateral plexuses in the lateral ventricles, and empty themselves into the straight sinus, and thence into the torcular Herophili and through



Figs. 154 and 155.—The upper figure shows the areas of distribution of the CEREBRAL ARTERIES—median surface of the brain above, external aspect of the brain below.

the lateral sinus into the internal jugular vein. Pressure on or thrombosis of these veins produces distension of the lateral ventricles with fluid (hydrocephalus) if the foramen of Majendie be occluded.

The PECULIARITIES OF THE CEREBRAL CIRCULATION are as follows—(1) there are no anastomoses between the arteries of the cortex and those of the interior of the brain; there are no anastomoses between the several branches in the interior, and only very few between the several cortical branches, each of which supplies its own area. (2) There are no communications between the several branches of the vertebral, basilar, and cerebellar arteries, which supply the pons and bulb; but the superior, middle, and inferior cerebellar arteries communicate freely, hence the greater frequency of vascular lesions in the former as compared with the latter positions. (3) The cranium being a rigid box the quantity of blood in it is always the same, but the velocity varies considerably, and the velocity depends on the blood pressure in the carotids and vertebrals. The blood pressure in these varies inversely as the dilatation of the splanchnic area, and the rapidity of the cerebral circulation, it seems quite clear, is regulated mainly by the great reservoir in the splanchnic area. When this is dilated the general blood pressure is low and the circulation in the brain is slow; conversely, when the splanchnic area is contracted the general blood pressure is raised, and the circulation of the brain becomes more rapid. Dr. Leonard Hill maintains that there is no vaso-motor regulation of the cerebral arteries, a fact which is hard to believe in view of the large amount of involuntary muscular tissue present in these vessels. Professor Sherrington maintains that they have the same nerve supply as the other vessels. At any rate, high blood pressure in the carotids and elsewhere in the cerebral circulation is attended with rapid circulation in the brain (wakefulness); and, conversely, low blood pressure is attended with slow cerebral circulation, as in sleep.

§ 505. **The Sympathetic Nervous System** is almost as extensive and elaborate as the cerebro-spinal nervous system, with which it is intimately connected. The sympathetic system consists in the main of a double series of ganglia arranged along the ventral aspect of the vertebral column. In the abdomen these ganglia are massed and matted together into the cœliac plexuses and semilunar ganglia, which evidently constitute the chief centres, the "brain" as it were, of the sympathetic system. The sympathetic ganglia are connected by non-medullated fibres with one another, with the cranial nerves, with the anterior and posterior roots of the spinal cord, with all the arteries (around which they form plexuses), and with all the unstriped muscular tissue of the body, namely that of the arteries and that of the intestines and other viscera. The only named branches are the greater and lesser splanchnic nerves.

The main function of the sympathetic system appears to be to regulate the flow of blood through the arteries by means of their tunica media or muscular coat and thus to control and co-ordinate the various vascular areas one with another and regulate the nutrition going to the tissues and viscera. It has been shown (by Dr. Leonard Hill for example) that the cerebral circulation is mainly regulated by the contraction and dilatation of the vascular area (the splanchnic) within the abdomen. Stimulation of the sympathetic fibres to the heart accelerates that organ. This system also regulates the intestinal movements and therefore the alimentation of the body. It also regulates the secretion of various glands either directly or indirectly through the vessels which supply them. The pathological effects are most definitely recognised in the case of paralysis of the cervical sympathetic (§ 605). In the author's view the sympathetic system is also largely responsible for most of the phenomena of hysteria, many of those ascribed to neurasthenia and some of the other so-called functional diseases of the nervous system.

§ 506. **General Principles in Neuro-pathology.** (1) *Whenever the nutrition of a neuron is impaired, the peripheral endings of its processes (dendrites) are the first to undergo degeneration.* An illustration of this is seen in tabes, where the peripheral terminations of the muscle sense neuraxon in the muscle spindle is the first to undergo degeneration. This principle, which has a widespread application in neuro-pathology, is the outcome of the fact that the cell or neuron-body is the most essential and vital part of the neuron, and controls the nutrition of the other parts. Consequently, when the neuron as a whole suffers from want of nutrition or other cause of degeneration, the parts furthest from the cell first show the degenerative change.

(2) *Prolonged forced functioning of any nerve structure results in its atrophy; it also results in the predominance and over-powering of that structure by the surrounding tissues, and a final degeneration of the fatigued structure.* This is sometimes known as Edinger's theory, but it is a principle with which neurologists have long been familiar. A typical instance of it is seen in the tremor, spasm, and atrophic paralysis which accompany various occupation neuroses. It explains also the predominance of tabes in the male sex, who use their muscle sense neurons so much more than females, and it also explains the fact why tabes supervenes during the prime of manhood, at a time when the muscle sense neurons are exercised most.

(3) *Sclerosis is a consequence, not a cause, of the atrophy of the parenchymatous tissues* (nerve structures proper). This is a general pathological principle, which is applicable also to the nervous system. It was formerly thought that the occurrence of fibrous tissue in cirrhosis of the liver, or fibrosis of the kidney (contracted granular kidney) was the cause, by pressure, of the atrophy of the glandular cells, but modern pathology teaches the converse of this, and shows how true was the statement of Cohnheim, made about the year 1860, that "the mutual resistance of tissues to each other's encroachment, limits tissue growth normally. If you remove one tissue element—by degeneration or otherwise—the surrounding tissues (deprived of this opposition), tend to take on increased activity, growth, and proliferation." An application of this principle is seen also in tabes, where, as a consequence of the degeneration and atrophy of the muscle sense neurons, secondary sclerosis occurs of the posterior columns, in which the muscle sense neuraxons lie. It is seen in many other scleroses of the spinal cord.

(4) *Functions which are last acquired in the evolution of the nervous system, are the first to succumb to disease, and vice versâ.* One illustration of this principle is seen in mental disorders, in which the latest acquired faculties of the mind are those which most readily become disordered. Again, in aphasia (loss of memory for the signs of thought), the memory for printed or written signs is lost more often and sooner than the memory for spoken words.

PART A. SYMPTOMATOLOGY.

The most constant and CARDINAL SYMPTOMS of nervous diseases are referable either to the **mind** or the **muscular** system. The *subjective symptoms* met with in association with disorders of the nervous system are very numerous, but they can practically all be brought under one of five headings—**nervousness**, **pain** (including **headache**), **disordered sleep**, **vertigo**, and **disordered sensations**.

The **mental powers** are disturbed in greater or less degree in many diseases of the nervous system, particularly of the brain. Sometimes there is only inaptitude for mental work; sometimes there are transient disturbances of thought; at others there is stupor, mental apathy, or coma. Sometimes there is loss of speech or of memory, or some other faculty of the mind; sometimes there is restlessness, excitement or delirium; at others there is a more chronic perversion of the mind which amounts to insanity. A simple epitome of these various symptoms and their causes occupies 37 pages (pp. 932 to 969). In the mental symptoms which arise in certain inflammatory and other diseases of the brain it is useful to remember that delirium, convulsions and headache are evidences of *cerebral irritation*, whereas mental dulness, stupor, paralysis and coma are evidences of *cerebral compression* or intense toxæmia.

Motor defects are, after the various mental symptoms just referred to, the most common among nervous disorders, because the muscular system is almost entirely dependent on the nervous system. These defects will be considered seriatim under paralysis (of several different kinds), inco-ordination and disordered gait, muscular rigidity and tremor, and muscular wasting, in Parts B. and C.

§ 507. **Nervousness** is also a mental symptom which frequently guides us to disorders of the nervous system. In its colloquial sense it generally means "easily agitated"; the patient comes to us for instance because the least noise startles him or the least worry upsets him. (1) It is the leading and most constant symptom in *neurasthenia*, an asthenic state of the nervous system (§ 518) arising from gastro-intestinal or other toxæmia, malnutrition, fatigue, accident, or some emotional strain.[1] (2) *Hysteria* is the next most common cause of nervousness. But here we have an inherent emotional and vaso-motor instability which is manifested by "hysterics" and various other forms of attack.[2] (3) In a great many structural diseases of the nervous system the patient comes to us for what he calls nervousness, particularly in those which will be mentioned under the symptom tremor (§ 583). (4) Chronic alcoholism, morphinism and their effects are manifested by nervousness; and (5) Convalescence from severe illness and many other debilitated conditions are also attended by nervousness.

Pain is a subjective symptom of considerable frequency in all diseases, but not more in those of the nervous system than other systems unless the peripheral nerves are involved. We know but little about the psychology and pathology of pain, but a careful clinical observer may derive considerable help by investigating the four important qualities which I have many times mentioned (1) its position; (2) its character, whether throbbing, pricking, shooting, knife-like, dull, aching, etc.; (3) its degree; and (4) its constancy, *i.e.*, whether persistent or intermittent, or a combination of the two (paroxysmal or exacerbating). We should never allow ourselves, for the sake of time or trouble, or by pandering to the ignorance or whim of our patients, to treat pain simply as pain, by the administration of soporifics, hypnotics, and the like. Pain is a sure indication of abnormal structure or function, and it is our bounden duty to endeavour to trace out its *causal agency*, however difficult the task may be. The best method of investigating the cause of a neuralgia or pain in any given case is 1st, to examine the nerve apparently involved in the pain, and the *locality* around, for direct causes of irritation, and particularly any bony orifice through which the nerve passes; 2ndly, to seek for any *reflex cause* of irritation in disease of organs more or less distant, such as the teeth or the uterus; and 3rdly, to search for any general or *constitutional derangement* which may act as a predisposing or exciting cause, such as anaemia, rheumatism, gout, tubercle, or syphilis.

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[1] "Clin. Lects. on Neurasthenia," 2nd Edn., Glaisher & Co., London, 1902.
[2] *The Lancet*, June 1, 1902, and *Clin. Journal*, May 25, 1904.

PAIN and NEURALGIA in different parts of the body and limbs are discussed fully under Neuralgia (§ 598).

§ **508. Headache** (cephalalgia) is often met with in diseases of the nervous system, but it quite as often attends morbid conditions of some other, and particularly the vascular system. Its generalised distribution on both sides (a feature which distinguishes it from neuralgia of the 5th) suggests that it is due in most cases to disturbances of the circulation within or outside the cranium. Frontal, vertical, and occipital headache were believed by Dr. Hughlings Jackson[1] to indicate an abdominal, cerebral, and circulatory origin respectively, but the position of the pain is not, in my experience, much guide to its cause.

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[1] Quoted by Dr. J. S. Bristowe, "Principles and Practice of Medicine," 4th edition, London, 1882

The *causes* of headache are numerous, and do not admit of pathological classification, but inquiries should be directed to possible LOCAL, GENERAL, and REFLEX causes as in the case of pain and neuralgia.

(a) Among the following LOCAL CONDITIONS the first 5 cause continuous pain of some duration. (1) *Syphilitic disease of the cranium* is a frequent cause of continuous headache, and a marked feature of this headache is its nocturnal exacerbation and its associated tenderness. (2) Various *meningeal conditions*, acute or chronic, cause pain, and here syphilis again may play a leading part. In children tuberculous meningitis should always be suspected, and the temperature carefully taken. (3) *Intracranial tumours*, especially when affecting the meninges or cortex, may be known by the association of vertigo, occasional vomiting (especially when the headache is worst) and optic neuritis; localised tenderness over the seat of the lesion is sometimes observed. (4) *Ear disease* causes headache, and in such cases pressure over the mastoid cells often reveals tenderness. (5) It must also be remembered that disease of the *frontal sinuses*, which is usually secondary to nasal or post-nasal catarrh gives rise to dull, continuous headache. (6) The *supra-orbital branch* of the fifth nerve supplies the forehead, and neuralgia of this nerve necessarily produces frontal headache of a shooting and paroxysmal character. (7) *Excessive brain work* is frequently followed by a feeling of dull heavy weight on the vertex. (8) The wearing of hard and heavy hats.

(b) Among the CONSTITUTIONAL or GENERAL CONDITIONS the first seven causes mentioned below are due to toxic or hæmic conditions, and perhaps eight and nine also; all of them are apt to have periodic exacerbations. (1) *Chronic interstitial nephritis* should always be suspected in the aged, a suspicion which is confirmed when the patient frequently rises at night and passes large quantities of urine of low specific gravity. (2) *High arterial tension* is probably in operation in the foregoing, but from whatever cause arising is a very frequent source of headache. Indeed, it is generally the earliest and most pronounced symptom which attracts the patient's attention. (3) *Hepatic derangement* and the condition known as *lithæmia* give rise to what is sometimes called bilious headache. (4) *Chronic alcoholism, gout, rheumatism, syphilis,* and *plumbism* are also attended by headache, but it has no special characters. (5) *Ague,* though rarely seen now in this country, is attended by a severe frontal headache; hence the the term "brow ague." (6) *Pyrexia,* due to any cause, is usually accompanied by headache. (7) *Chlorotic patients* suffer a good deal from headache, but it has no special features beyond the concurrent signs of anaemia. (8) Closely allied with the preceding is the headache of *exhaustion* or inanition. (9) *Hysteria* and *neurasthenia* are frequently attended by headache; the first is often likened to a nail being driven into the skull at one spot (hence the term "clavus" applied to this headache); the second very frequently takes the form of a feeling of constriction around the head ("casque neurasthenique" of Charcot). (10) *Migraine,* which is a special paroxysmal form of headache, will be described below (§ 599).

(c) REFLEX CAUSES. (1) *Asthenopia* or eye strain is an extremely frequent cause of headache in modern times, and is generally associated with some uncorrected error of refraction or astigmatism. It may arise without any error in those who read small print too much. This kind of headache has, in my experience, fairly constant characters, viz., it is worst on rising in the morning, improves a little, and then again gets worse after the day's work. (2) *Constipation.* (3) *Dyspepsia* and derangement of the stomach are certainly amongst the most frequent causes of headache in everyday life. It is said by some to be situated in the frontal region: but in my belief it has no special characters beyond the associated symptoms of these maladies. (4) In *diseases of other viscera,* especially of the heart, lungs, uterus, and liver, a pain referred to the head is often present. The headache of heart disease is often known as congestive headache, and is certainly best relieved by cardiac depressants or bleeding.

The palliative *treatment* of headache as a symptom will be found under neuralgia and migraine.

§ 509. **Disordered sleep** is connected mainly but not exclusively with nervous disorders. We have to rely very much upon a patient's own account, and some care is required in accepting his statement in this matter. Persons differ considerably in the amount of sleep they require. The aged will do with half the sleep of adolescence, and the middle-aged with half the sleep of babyhood, the respective quantities being approximately 5 and 10 hours, 8 and 16 hours. Brain workers and town dwellers require more sleep than labourers and country folk, though as a rule they get less. The popular belief that "6 hours for a man, 7 for a a woman, and 8 for a fool" is sufficient sleep, is highly fallacious. Sir William Jones' adaptation in the 18th century of an old Persian saying—

> "Seven hours to work, to soothing slumber seven,
> Ten to the world allot, and all to Heaven,"

comes much nearer the truth, and my own belief is that a town life with brain work requires at least 7 or 8 hours of quiet and uninterrupted sleep.

Sleep may be (a) DEFICIENT in quantity (insomnia), or (b) DEFECTIVE in quality (restlessness, dreaming, etc.); these generally co-exist and their causes are interchangeable. (c) It may be EXCESSIVE in quantity. Sleep is attended by anæmia of the brain. The causes of (a) and (b) depend either on inherent irritability of the brain, or an excess or defect in quality of its blood supply.

(a) INSOMNIA, wakefulness, or deficient sleep may arise under the following conditions. (1) All *painful affections* are apt to be attended by sleeplessness. (2) *Defective hygienic conditions* or mode of life, *e.g.*, late hours, late suppers to those unaccustomed to them, indigestible food, overwork of mind or body, unaccustomed surroundings and the like. (3) In neurasthenia, hysteria and other functional *disorders of the nervous system*, mental excitement, all acute and many chronic forms of mental derangement, sleeplessness may be one symptom of the ailment; or its cause. (4) In the absence of any of the foregoing causes, search should be made for some *general constitutional condition*. Sleeplessness, like headache, in the aged should always make one suspect chronic interstitial nephritis, and such patients often complain of "cat-sleeps," *i.e.*, dropping off for a few minutes at a time. (5) Among *local disorders*, cardiac valvular disease may be mentioned, in which the patient starts up as soon as he falls off to sleep with a feeling of suffocation.

(b) DEFECTIVE SLEEP. (1) *Dreams* and nightmares occur in many of the disorders above named. In the young they are more often due to an undigested meal taken late. They are also a characteristic symptom of neurasthenia, anæmia, deficient oxygenation, and various other toxic conditions of the blood. (2) *Night terrors* in children are sometimes induced by worms or late suppers, or other dietetic or gastro-intestinal defects especially in nervous children. Among other causes may be mentioned nasal or pharyngeal obstruction (*e.g.*, adenoids). If continual they should make us suspect petit mal, especially when combined with nocturnal incontinence. There is a residue of cases in which no cause is apparent excepting the neurotic diathesis, which subsequently becomes manifest by the development of hysteria, epilepsy, neurasthenia, or chorea.[1] (3) *Sleep-walking* and sleep-talking are curious phenomena in which certain functions of the brain are not only awake, but sometimes in an exalted condition. They are allied to hysteria, and the same treatment applies. Here again petit mal may be in operation. (4) *Twitching of the limbs* as a person drops off to sleep may be an early symptom of peripheral neuritis, or of neurasthenia. It is a common symptom of some toxæmias (particularly those of intestinal origin) on the one hand, or hypersensitiveness of the nervous system (such as occurs in morphinism, § 521) on the other. In all of these the twitchings may pass on to intolerable restlessness.

(c) EXCESSIVE SLEEP. Drowsiness is a symptom met with in the intense venous congestion of *heart disease*; it is also an early symptom of *acute uræmia*. In some other *toxic states*, and some *organic brain* affections (especially tumour in the region of the third ventricle), sleepiness is met with, and in all of these it tends to pass into stupor (§ 525). But sometimes it is met with in apparent health. I was once consulted by a curate who went to sleep almost as soon as he sat down in church during certain parts of the service, or when his rector was preaching—a most inconvenient circumstance. In that case it was apparently due to *lithæmia* and *disordered liver*, which being remedied, the sleepiness disappeared. Dyspepsia (when the somnolence follows meals) and anæmia may act in the same way. The *trypanosoma*, when affecting the cerebro-spinal fluid, produces "sleeping sickness," in which the patient sleeps for days and then generally dies. Persistent and even fatal sleep has been known to follow *influenza*.

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[1] See also a clinical lecture by Dr. Leonard Guthrie, *Clinical Journal*, June 7, 1899.

The *treatment* of insomnia must be directed to the cause. It is in cases of sleeplessness with or without pain that hypnotism is sometimes of value. Among the simpler remedies, I have often found useful a cup of warm milk or gruel, or a hot bath or hot-pack, last thing at night. Sometimes an evening walk is useful, or Swedish and deep breathing exercises, or some means which take the mind away from the occupations of the day. Massage or cold compresses over the legs or abdomen may promote sleep. Sod. hypophosphite grs. xx. in warm milk or milk and water at bed-time is a simple yet excellent remedy for the sleeplessness of mental fatigue, or bromide of ammonium 20 grs. thrice daily, or an alkaline draught of 60 grains of bicarbonate of soda in a tumbler of hot water. A weak galvanic current I have sometimes found good. When such simpler measures are unavailing, recourse must be had to hypnotics and sedatives—care being taken not to engender a habit—such as the following: alcohol, chloral hydrate, camphor, cannabis indica, codeina, hyoscyamine, lupulin, narceine, paraldehyde, bromidia, chloralamide, chloralimide, chlorobrom, hypnal, sulphonal, tetronal, trional, urethane, chloralose, scopolaminæ hydrobromide, chloretone.

§ 510. **Vertigo** is a subjective sensation experienced by a patient that either he or the objects round him appear to be rotating, accompanied by a momentary loss of equilibrium. The latter may be so marked as to lead to a reeling in the gait, or actual falling down. The symptom is sometimes difficult to elicit without putting a leading question, but it is best to ask the patient if he has "any sensation" in the head. People describe it sometimes as a "giddiness," a "dizziness," or "swimming in the head." The rotation may appear to be horizontal, vertical, or oblique. The sensation is often attended by nausea; and intense giddiness, from no matter what origin, may be sufficient to cause vomiting.

The *causes* of vertigo may be mentioned under 6 headings—(a) diseases of the ear, (b) diseases of the nervous system, (c) diseases of the eye, (d) diseases of the circulatory organs, (e) affections of the stomach (gastric vertigo), and (f) laryngeal vertigo. The first thing to do in any given case of vertigo is to decide whether it be accompanied by any defect of hearing or not, as being the commonest cause.

(a) AUDITORY OR AURAL VERTIGO. Any disease of the external meatus, Eustachian tube, or middle ear, which is accompanied by alteration of pressure on the fenestra ovale, is usually accompanied by giddiness and almost invariably by defective hearing. The deafness may be either *obstructive deafness*, such as arises in chronic middle ear catarrh, or *nerve deafness*, such as that in labyrinthine disease. These conditions are mentioned as the commonest in the two classes named, but there are many other less common forms of obstructive deafness and nerve deafness which are attended by vertigo (§ 614).

(b) DISEASES OF THE NERVOUS SYSTEM. (1) In *epilepsy*, vertigo often constitutes the aura or warning of convulsive attacks (grand mal) and the whole phenomenon in slight attacks of minor epilepsy (petit mal). It may be known by being invariably accompanied by a temporary interruption of consciousness, however slight the attack, by a history of other attacks dating from about 14 to 20 years of age, and by the absence of signs of gross lesion. (2) *Hysteria*, *neurasthenia*, or any exhausted or depressed condition of the nervous system, such as that produced by drugs, alcohol, or mental strain, may be accompanied by giddiness. Hysterical vertigo generally comes on only when the patient is startled or frightened. Neurasthenic vertigo is usually apt to come on when the patient goes out of doors. (3) *Various intracranial lesions* in different situations may give rise to giddiness. Intracranial tumours in general, but especially those affecting the cortex, cause vertigo and are usually also accompanied by headache, vomiting, optic neuritis, and perhaps convulsions, together with paralysis of some of the cranial nerves. Vertigo is a very marked feature in lesions of the *cerebellum* or its peduncles, and here giddiness and a reeling gait are often the leading and sometimes the only symptoms. (4) *Disseminated sclerosis* is attended in three-fourths of the cases (Charcot) by vertigo. A disease has been described under the name *endemic paralytic vertigo*, occurring in Switzerland and Japan, characterised by paroxysmal vertigo and paralysis.

(c) In regard to CIRCULATORY DISORDERS, (1) giddiness constitutes the first stage of syncope, which may stop short at vertigo. In this way any condition of debility or poverty of blood, anæmia, convalescence, or exhausted states of the system, may give rise to giddiness. Anæmia is perhaps the commonest cause of vertigo in the young. (2) Any *cardiac weakness* or degeneration, or disease of the coronary arteries, may give rise to vertigo, especially among the aged. Excessive smoking, by acting as a cardiac depressant, may act in the same way. (3) *Arterial disease* is attended by vertigo, which even more than the preceding is characterised by coming on chiefly when the patient rises from a sitting or stooping posture. It is as I have described elsewhere[1] a true postural vertigo and very often occurs on rising at night to pass water or on first getting up in the morning. (4) Vertigo in the aged may also be the only indication at the time of their occurrence of the *minute hæmorrhages* or *softenings* so often found in their brains after death. (5) *High arterial tension* arising from various permanent or temporary causes, as, for example, excess of uric acid in the blood, is not infrequently attended by slight giddiness. Apart from the actual occurrence of hæmorrhage, vertigo in the aged may always be taken as a warning of some kind of cardiac or vascular failure; and the circulatory system should be carefully investigated.

(d) OCULAR VERTIGO is usually due to weakness of one or other of the ocular muscles. It is not uncommon in cases of myopia, in which the constant strain of the internal recti leads to weakness of these muscles. It may also be produced by other ocular conditions, especially in diplopia from any cause. Here, the vertigo is characterised by being relieved by shutting one or both eyes. So also is the dizziness felt on climbing a height, or being in wide open spaces as in the agoraphobia of neurasthenia.

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[1] *British Medical Journal*, Jan. 23, 1897, and Transactions of the Pathological Society of London, 1904.

(e) GASTRIC VERTIGO is a variety which has been described by several observers (*e.g.*, by Trousseau), but for my own part I believe many of such cases are of circulatory origin. The fact of the close relation between the taking of food and the giddiness, to which some refer, is sufficiently explained by the effect of a full stomach upon the abdominal sympathetic or mechanical impediment to the heart. Disorder of the liver is also credited with producing giddiness.

(f) A LARYNGEAL VERTIGO has been described in which the patient gets giddy and may fall on attempting to cough.

The *treatment* of vertigo must be directed to the cause, but as a palliative measure the bromides are of great value.

Disordered subjective sensations of many kinds may be complained of in diseases of the nervous system. They can be grouped more or less into four categories—

a. Subjective sensations referable to the *extremities* such as numbness, tingling, etc., which will be mentioned under peripheral neuritis and disorders of sensation.

b. Subjective sensations referable to the *special senses*, smell, sight, hearing, and taste, which will be mentioned under diseases of the cranial nerves.

c. Subjective sensations referable to the *mind*, hallucinations, delusions, etc., which are dealt with in mental disorders.

d. Subjective sensations which affect the *equilibrium* are mostly included under the term vertigo or giddiness (*vide supra*).

PART B. CLINICAL INVESTIGATION.

§ 511. The method of examining a nerve case differs somewhat from that in other departments of medicine partly on account of the inaccessibility of the nervous system to direct examination, and partly owing to the widespread extent and indirect effects of disease. It is, however, not difficult provided the beginner adopts a fixed order of examination. It requires a little more time, but if the reader studies carefully what follows he should find no more difficulty in diagnosing diseases of the nervous system than those of the heart or stomach; especially if he remembers that there are two steps in the diagnosis of nervous cases—First, to LOCALISE the lesion (is it generalised, or in the brain, the cord, or the nerves?), and secondly, to ascertain what is the NATURE of the lesion. The following table represents a scheme which may be adopted for a systematic and complete investigation, taking first that nervous function to which the patient's symptoms are mainly referable.

First, the leading symptom or sign.

Secondly, the history of present illness, previous, and family histories.

Thirdly, proceed to the examination of the—

I. Mental and general symptoms which first attract attention, including any defect in the skull. The temperature in certain cases.
II. Muscular system—weakness, walk, spasm, tremor, atrophy
III. Reflexes—deep, superficial, or organic (sphincters).
IV. Cutaneous sensation and pain.
V. Special senses and cranial nerves.
VI. Trophic changes—bedsores and trophoneuroses.
VII. Electrical reactions—if necessary.

The **history** of the case is of considerable importance in diseases of the nervous system and must be patiently and thoroughly investigated.

The *history of the present illness* needs careful inquiry, because so many nervous symptoms are vague or what the patient regards as unimportant. Sometimes the onset is acute, but far more frequently it is most insidious and chronic. Paraplegia and many other diseases run a very prolonged and changing course and the physician who sees a case 3 or 4 years after its onset may be wholly without reliable data on which to found a diagnosis as to the nature of the lesion, other than that which the history reveals.

The *previous history* may reveal lead or other metallic poisoning, gout, tuberculosis, syphilis, or other predisposing causes of nervous affections; syphilis may also be a determining factor. Alcohol and syphilis play a most prominent part in the etiology of nearly all diseases of the nervous system. The toxins of diphtheria and influenza among the infective fevers seem to be the most inimical to the nervous system. Traumatism, mental and emotional strains often play a part in the etiology. The influence of sexual abuses is considerable, but is often in my belief exaggerated. Some diseases like hysteria and migraine are recurrent throughout life and the history is a most important aid to diagnosis.

In the *family history* a neuropathic diathesis may be revealed by ancestors and relatives having suffered from some nervous disease. The more important inherited diseases are epilepsy, hysteria, mental disease, migraine and other vaso-motor conditions. Consanguineous marriages intensify and sometimes initiate this diathesis.

§ 512. I. Mental and general symptoms. The majority of cases met with in practice and out-patient work belong to the **generalised neuroses**, where the symptoms consist of vague bodily discomfort, nervousness, restlessness, etc., localising signs being absent. In such cases an *examination of all the organs* *and functions of the body* will frequently reveal some defect to which the nervous condition is secondary, and such an examination should not be omitted.

The **mind** of the patient plays a very important part in all disorders of the nervous system. We have to judge how much of the malady exists in the patient's mind, how far we can rely on his description of his symptoms, even apart from any mental or moral alienation which may exist. Careful observation during our investigation of his medical history may teach us much concerning a patient's mental condition, and some of the chief points to investigate are—sleep, dreams, memory, intelligence, reasoning power, decision, attention, moral and ethical standards, delusions, hallucinations. In judging a person's mental condition or ability one must be a well-read man of the world first and a physician afterwards. This was what Sydenham meant when he replied to a gentleman who asked what books his son should read as a preparation for the medical profession—"Let him read 'Don Quixote,' that's a very good book." Common sense and tact, as well as ability and experience, are indispensable. It is, moreover, necessary to enter sympathetically into the feelings and thoughts, mode of life and mental attitude of your patient before he will favour you with his entire confidence.

The investigation of the **skull** is considered in § 13 and § 620.

Pyrexia is not often present in diseases of the nervous system. Moreover, relatively very few lesions (besides vascular lesions of the nerve structures) come on suddenly, and even in these the temperature is seldom elevated. Even in cerebral abscess pyrexia may be absent excepting just at the outset. Practically, persistent fever in a case of nervous disease indicates some inflammation of the cerebro-spinal meninges.

§ 513. II. The **muscular system** is the part next most liable to give evidence of a nervous lesion. The muscles may be affected in 4 ways—

(a) WEAKNESS or paralysis.
(b) Alteration of GAIT or INCO-ORDINATION.
(c) Muscular SPASM, tonic or clonic, or tremor.
(d) ATROPHY.

There are two other points which are specially related to muscular defects:—the condition of the deep, superficial, and organic REFLEXES; and the ELECTRICAL REACTIONS of the muscles involved.

(a) Is there any **loss of power**? Can the patient walk? Can he sit up in bed? Can he move each of his limbs as a whole? Is the motor weakness localised to a few muscles? These are all points to investigate. The *degree* and exact *position* of the weakness (which enables us to localise the lesion in the nervous system) should then be investigated. Paralysis is a total, paresis a partial, loss of power. In the case of the hand-grip the degree of weakness can be measured by a dynamometer, but in other instances we can only roughly estimate the degree of weakness of a muscle or group of muscles by the strength of passive resistance on the part of the operator required to prevent a certain movement.

HEMIPLEGIA is paralysis of one side of the body; PARAPLEGIA, paralysis of both legs; MONOPLEGIA, paralysis of one limb (crural of one leg, brachial of one arm); BRACHIPLEGIA, paralysis of both arms; DIPLEGIA, paralysis of both sides of the body; DIPLEGIA FACIALIS, both sides of the face. In cases of apoplexy or coma it is often difficult to test the presence of paralysis, but its existence on one side may be indicated by a greater limpness on that side—the arm when raised and allowed to drop will fall inertly, or it can only with difficulty be raised because of paralytic rigidity. Sometimes an *individual muscle* or a *group of muscles* is affected and a knowledge of the action of muscles enables us to decide which is involved. Go patiently through the movements of a joint or those performed by individual muscles, *offering passive resistance* to each movement in turn; this reveals the position and the degree of the defect. The big joints are capable of 6 movements—flexion, extension, adduction, abduction, rotation and circumduction; the smaller joints only the first two or the first four. The action and nerve supply of the various muscles are given under plexus and single nerve paralysis (§ 564).

(b) Is there any **alteration of gait** or **inco-ordination**? A patient should if possible be made to walk before us. The *gait* in locomotor ataxy, paralysis agitans, spastic paraplegia, and many other affections is very characteristic (§ 571).

INCO-ORDINATION is a defective co-operation of the different muscles involved in a particular movement of a limb, unaccompanied, it may be, by any loss of muscular power. In the *legs* inco-ordination is apparent by an exaggeration of the normal movements (as in locomotor ataxy) or an inability to balance while walking (as in cerebellar tumour). The patient may be asked to walk along the edge of the carpet. If the patient is in bed ask him to follow your finger round in a circle with his big toe, or to touch with his eyes closed the dorsum of one foot with the big toe of the other. Another test is to ask him to stand with heels together and the eyes shut and notice if he stands steadily or sways about (ROMBERG'S SIGN). A very delicate test of the same kind is to ask him to balance himself on tiptoe with knees bent and eyes closed. To test the *upper extremities* ask him to thread a needle, or (with eyes shut) bring his 2 forefingers tip to tip in front of him, or to touch the tip of his nose. Co-ordinated muscular movement depends upon the integrity of the muscle sense, the vision, and the cerebellar control. The eyes are closed or bandaged when testing in order to eliminate the vision.

Muscle Sense is the power of appreciating or judging the state of contraction of a muscle. It is an extremely delicate sense, for it is mainly by the muscle sense of the ocular muscles that we judge distance. In tabes dorsalis this sense is defective. It is tested in two ways—(i.) The *sense of weight* is tested in the arm by placing objects of the same size and configuration but of different weights in the patient's hand. A match-box with coins and one with cotton wool inside or balls of same size but different weights may be used; or the objects can be placed in a handkerchief and slung on to the hand or foot. Normally a healthy person can detect a difference of one-seventeenth between two weights. (ii.) The sense of *position* of a limb is tested by bending a joint about in various positions and finally asking the patient in what position you have left the fingers or limb—bent or straight—or asking him to put the other hand or leg into the same position. The operator must cease to touch the skin and the finger or toe must not be in contact with any other member or object, otherwise the patient's tactile sense comes into play. Another rough test is to tell the patient with his eyes closed to touch the tip of his nose with the tip of his first finger.

(c) Is there any **spasm, tonic** or **clonic,** or **tremor**; if so, note its kind, degree, and distribution. Spasm or involuntary muscular contraction may be of two kinds—*tonic* when the contraction is continuous, *clonic* when it is intermittent; smaller, more rapid or vibratory movements are known as *tremors.*

A *tonic* rigidity is obvious on attempting to bend the limb: it may be generalised, as in the case of tetanus, or localised to the paralysed limbs in hemiplegia and paraplegia (owing to descending sclerosis). Early and late rigidity are referred to under hemiplegia.

Clonic spasms such as occur in chorea, and *tremors* or smaller movements such as occur in paralysis agitans are equally obvious. Tremors are rendered more obvious by the patient holding up the affected member. It is important to note as hereafter explained whether these movements are present only during muscular action (intention tremor) or not.

Athetosis is a peculiar condition of slow mobile spasm intermediate between tonic and clonic spasm.

Convulsions are violent clonic spasms. It is important to ascertain—1st, the point at which the convulsions started or predominated; or whether they were generalised; 2ndly, whether the patient was unconscious or not; 3rdly, whether the fit was preceded by a warning; 4thly, what was his condition after the fit; 5thly, whether there was any involuntary evacuation, or biting of the tongue; 6thly, whether the patient has had any previous attacks of the same or a different kind.

Hypertonia or hypertonicity is increase in the tone of all the muscles hardly amounting to tonic spasm; *hypotonia* is a diminution of the muscular tone. *Kernig's sign* which is found in 85 per cent. of cases of acute cerebro-spinal meningitis is an evidence of hypertonicity. It consists of a strong contraction of the hamstring muscles after they have been stretched. To elicit it with the patient in the recumbent posture, bring the thigh to a right angle with the abdomen; then on trying to extend the leg on the thigh strong contraction of the flexors prevents full extension.

(d) Is there any **muscular atrophy**? Atrophy may be elicited roughly by pinching the muscles and finding them flabby and wasted, or by the measuring tape. Finer defects of muscular nutrition can only be obtained by electrical examination (p. 914).

Muscular atrophy arises from (1) *disuse*; (2) diseases of the *spinal cord* which involve the anterior horns; (3) diseases of the *peripheral nerves*; (4) diseases of the *muscles* (myopathies); and (5) diseases of the joints.

§ 514. III. There are three kinds of **reflexes** to be investigated —**deep, superficial,** and **organic.**

(a) Are the **deep reflexes** altered? When the tendon of a muscle that has been put on the stretch is struck the muscle immediately contracts and produces a jerk of the limb. This is known as the deep or "tendon reflex." To elicit the KNEE-JERK or PATELLAR TENDON REFLEX get the patient if possible to sit on the edge of a bed, table or chair, with the legs *hanging freely*, or cross one leg over the other and let it hang as though it did not belong to him. These positions slightly stretch the quadriceps extensor and reflexly increase its tone. Now strike the patellar tendon sharply with the tip of the fingers, or the edge of a rubber shod hammer or stethoscope, and the leg will immediately jerk forwards from the sudden contraction of the quadriceps. The patient's attention may be engaged by conversation or by hooking the fingers of the 2 hands tightly together, and trying to pull them apart. By some observers this is called the "reinforcement" of the knee-jerk. The strength of the knee-jerk varies in health; it is less marked in the young and in the old. An increase in the knee-jerk may be conveniently indicated by k.j. + 1, 2, or 3. It is exaggerated in all **upper motor neuron** lesions; it may also be increased in hysterical and other functional neuro-muscular irritability. It is diminished or lost in lower motor neuron lesions or whenever the reflex arc is interrupted by disease, as in locomotor ataxy.

The *knee-jerk is increased* (k.j. +), (1) When the lateral columns are affected by sclerosis, after cerebral or spinal lesions (sooner or later *all upper neuron lesions* are so attended). (2) When there is increased irritability of some part of the reflex arc, as in (i.) tetanus and strychnine poisoning, or (ii.) spinal meningitis. (3) When there is defective inhibitory control from the higher centres, as in hysteria (strychninism of Charcot) or in exhausted states such as phthisis, typhoid or neurasthenia.

The *knee-jerk is diminished* (k.j. —) *or absent:* (1) In all lesions of the lower motor neuron, such as infantile paralysis and other diseases of the anterior horns, and peripheral neuritis (alcoholic, diphtheritic, etc.). (2) In certain chronic spinal lesions, notably tabes dorsalis, in some cases of disseminated sclerosis when the grey matter is involved, and most cases of syringomyelia. (3) In certain acute lesions of the cord other than infantile paralysis: thus, in myelitis affecting the lumbar enlargement, and in transverse myelitis or other complete transverse lesions of the spinal cord the knee-jerk at first is generally, but not always, absent. (4) In primitive myopathies (idiopathic myopathy and pseudo-hypertrophic paralysis), in proportion to the loss of muscular power. (5) In cerebellar tumour, especially in tumours of the lateral lobe. (6) During coma, and also directly after the convulsive stage of epilepsy.

Most of the *superficial tendons* can be tested in the same way, though not with the same facility, the points being (1) to get the muscle to be tested relaxed by the patient, (2) gently stretch the muscle, and (3) strike its tendon. The tendo Achillis or *ankle-jerk* may be tested while the patient is kneeling on a chair with the calf relaxed, the foot being bent forwards and the tendon struck by the operator. The *triceps* or *elbow-jerk* is elicited by hanging the patient's elbow over your wrist and striking the triceps tendon ; the *supinator jerk* by tapping the tendon just above the styloid process; and the *wrist-jerk* by striking the extensor tendons when the hand is hanging loosely. The *jaw-jerk* is not present in health ; when present it can be elicited by placing one of your fingers firmly on the front of the chin and tapping it with the other as in percussion.

Ankle-clonus or the clonus imparted to the calf muscles by stretching the tendo Achillis, is elicited by supporting the patient's knee with one hand and suddenly dorsiflexing the foot with the other hand, gently maintaining the pressure of your hand on the sole all the time. It is present *with organic diseases* in the same circumstances as increased knee-jerk and ankle-jerk, *i.e.*, in *upper neuron lesions*. Under the same conditions knee clonus may be obtained by placing one finger above the patella, the patient being in a recumbent position, and percussing the finger with the other hand.

The explanation of deep or tendon reflexes is not yet quite certain. The phenomenon is too rapid for a reflex action, but for clinical and practical purposes it depends upon the integrity of a reflex arc, namely—(1) the sensory or afferent tract running from a tendon; (2) the ganglionic cells in the grey matter of the cord at a given level, and (3) the efferent or motor path from cord to muscle. If any of these are diseased (as they are in all lower motor neuron lesions) the corresponding jerk is absent. On the other hand, in upper motor neuron lesions, owing, it is assumed, to the cutting off of the habitual cerebral inhibitory influences, the jerks are exaggerated.

The tendon reflexes are of use not only to detect which of these two important groups of lesions is present—upper or lower motor-neuron lesions—but also to ascertain approximately the level of the cord which is involved, as may be seen from the table below.

Table showing the Spinal Segment involved in the Deep Reflexes.

Reflex.	Spinal segment involved.	Opposite vertebral spines of
K.-J.	2nd and 3rd L.	10 and 11 D. spines.
Ankle-clonus.	3rd, 4th, and 5th Sac.	1st L.
Elbow-jerk.	7th cervical.	4th C. spine.
Supinator-jerk.	5th cervical.	3rd C. spine.
Wrist-jerk.	6th cervical.	4th C. spine.
Jaw-jerk.	Motor nucleus of the 5th cranial nerve.	——

Myotatic Irritability consists of the too-ready contraction of a muscle when its muscular substance is struck. This is frequently seen in advanced phthisis, and other exhaustive diseases, *e.g.*, in percussing the chest. In tetany the condition is very marked, the facial muscles being thrown into contraction when struck or scratched.

(b) The **superficial reflexes.** On stimulation of certain parts of the skin or mucous membrane with a blunt pin or the top of a penholder, a contraction of certain associated muscles takes place, as shown in table in § 551. It is a true reflex action, and can only be obtained when the afferent and efferent paths and the corresponding grey matter in the cord or brain are intact. The chief use of the superficial reflexes in medicine is to determine the locality of a disease in the spinal cord. The mode of eliciting and situation of the ganglionic centre for the plantar, gluteal, cremasteric, epigastric, abdominal and interscapular reflexes are given in the table just referred to. The conjunctival reflex, obtained by touching the conjunctiva, causes contraction of the orbicularis palpebrarum; and its ganglionic centres are situated in the 5th nucleus (sensory) and the 7th nucleus (motor). The palate reflex, obtained by touching the soft palate, leads to its elevation by the levator palati; the afferent nerve is the 9th, the efferent the accessory part of the 11th (through the vagus). The cutaneous reflexes vary considerably in different individuals. They are normally more prompt in children than adults, and in women than men, and are difficult to obtain when the skin is harsh and insensitive as it usually is in the aged or when there is much subcutaneous fat.

Babinski's Reflex is a modification of the plantar superficial reflex. To elicit it the patient should be in the recumbent posture, with the lower limb slightly flexed and the sole of the foot warm and dry. The sole is gently stroked upwards by the finger-nail or a blunt pin. In health the big toe and the other toes will become flexed upon the sole: this is the normal reaction. In Babinski's reflex, there is *extension of the great toe* followed by flexion of the other toes. The stroking should not be hard enough to evoke dorsi-flexion of the foot, as this obscures the big toe reflex. This reaction is only met with in *organic disease* involving the lateral column. Babinski found it was elicited best by stroking the outer side of the sole. In *functional* cases the plantar reflex, if it can be elicited, gives a *flexor* response as in health (in functional cases a diminution of the plantar reflexes with exaggerated knee-jerks is a characteristic combination), and the same obtains in peripheral neuritis, poliomyelitis, tabes, and even in intracranial tumours, provided the pyramidal tracts are not involved. In infancy, before the age of walking, an extensor (not a flexor) response is normal.

(c) **Organic Reflexes.** Defæcation, micturition, deglutition, and respiration are sometimes spoken of as the organic reflexes, because they consist of the contraction of involuntary muscular tissue, resulting from the automatic stimulation of a mucous membrane. As regards *defæcation* and *micturition*, the patient should be questioned as to the existence of retention or incontinence, particularly in cases of paraplegia. The presence of such symptoms indicates direct or indirect involvement of the lumbar enlargement, and frequently occurs in myelitis and certain system lesions such as tabes dorsalis.

§ 515. IV. **Cutaneous Sensation.** Three kinds of cutaneous sensation are now recognised by authors—touch, pain, and thermal sense—and the two chief points about each of these are whether it is increased or diminished, and what the boundaries are of such alteration. To test a person's capacity to feel we must first obtain his intelligent co-operation, honesty and good will being large factors in the investigation. His eyes should be bandaged and he should be instructed to say simply "yes," immediately he perceives any sensation. Corresponding points on the opposite sides of the body should be tested if possible and a negative test should be applied from time to time. The sensibility differs in different persons and in different parts of the body.

(1) Cutaneous sensibility for TOUCH may be tested by the smooth head of a lady's hat-pin, the finger, the corner of the handkerchief, or if desired to altogether eliminate the sense of pressure, a small pad of cotton wool. For delicate investigations an æsthesiometer is sometimes used. Ascertain whether (i.) sensation is *lost* (anæsthesia) or *increased* (hyperæsthesia), and (ii.) what are the *boundaries* of such loss or increase (*e.g.*, hemi-anæsthesia or hemi-hyperæsthesia). Hysteria and locomotor ataxy are frequent causes of altered sensation. In the former, spots of an an- or hyper-æsthesia may be present, and pressure on the tender spots (hysterogenic zones) may produce fits. (iii.) Tactile sensation may be *delayed*, as in peripheral neuritis, or (iv.) *misplaced* (allocheiria) as in tabes. (v.) The *pressure sense* is really an element in the tactile cutaneous sense and has no known clinical importance apart from it; but if the student has a sovereign he can try if the patient can recognise the difference between this and a shilling placed on the skin when *the limb is supported* and at rest.

(2) Cutaneous sensibility to PAIN may be tested by the sharp end of the hat pin or by a faradic current. Occasionally, without loss of tactile sense, cutaneous sensibility to pain is lost (analgesia), or the reverse (hyperalgesia).

(3) Cutaneous sensibility to TEMPERATURE (thermal sense) is tested by test tubes of hot and cold water, or hot and cold silver spoons, the patient being asked to identify them. Loss of the thermal sense is a constant feature of syringomyelia.

The **sensibility of the muscles and nerves** to pinching and deep seated pressure should be tested. Both are very tender in peripheral neuritis. "MUSCLE SENSE" is one of the functions of co-ordination, and is described in § 513. **Arthritic sensibility** to torsion and **sensibility of the mucous membranes** are lost in some cases of hysteria. **Paræsthesia** (perverted sensation) and various subjective sensations are described in the disorders of sensation. **Aura** is a term applied to a morbid sensation or "warning" which precedes epileptic fits.

Pain and its method of investigation is mentioned under neuralgia, and in various other parts of this work.

V. The investigation of the **special senses** and **cranial nerves** is given in detail hereafter (§ 602). The points to investigate in order summarily given are as follows, the Roman numeral referring to the cranial nerve involved—

Symptoms referable to the nose—smell (I.); discharge.

Symptoms referable to the eye—vision, ocular movements, pupils, and fundi (II., III., IV., and VI.).

Taste, facial sensation and mastication (V.).

Facial movements (VII.).

Hearing, tinnitus, or vertigo (VIII.).

Pharyngeal sensation, deglutition, muscles of palate and larynx; also sterno-mastoid and trapezius (IX., X., XI.).

Muscular power of tongue (XII.).

VI. **Trophic Changes.** The nervous system controls the nutrition not only of the muscles, but of all the organs and tissues of the body. The trophic change which results may be revealed in a variety of ways.

The *skin* may become "glossy," *i.e.*, smooth, thin and atrophied. Erythema, ulceration (as in the perforating ulcer of tabes) or sloughing (*e.g.*, bedsores) may appear, vesicles may develop when an irritative lesion of a ganglion (*e.g.*, of the 5th cranial nerve) is present, and painless whitlows on the fingers, as in syringomyelia or Morvan's disease.

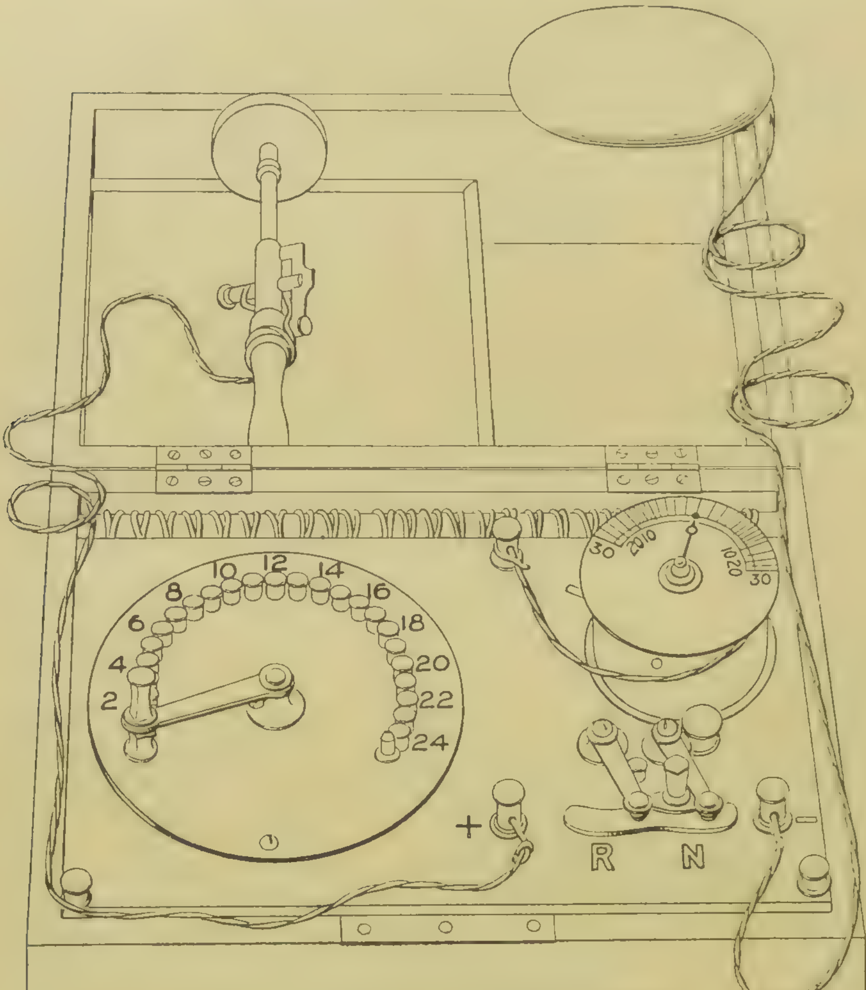

Fig. 156.—GALVANIC BATTERY with accessories necessary for electrical testing and treatment.

The *bones* may undergo absorption or arrested development (as in poliomyelitis).

The *joints* may be the seat of painless effusion and absorption of the bone ends (as in tabetic arthropathy).

§ 516. VII. **Electrical examination of muscles** and the nerves which supply them is an important aid in distinguishing lower from upper motor neuron lesions, and in detecting the degree of disease or degeneration in a nerve or muscle.

The APPARATUS REQUIRED is not complex. The chief requisite is a *faradic coil*, because the first and most important point is to ascertain the degree of faradic contraction. A simple Ruhmkorff coil, of which the secondary coil slides on to the primary is needed. It is better if the wire of the former is not too thin, as it produces unnecessary pain. A couple of moderate sized Leclanché cells will drive it. Two wires and three electrodes are required, one large (measuring about six by four inches), a round medium-sized one, and one small (about one inch in diameter). The two smaller ones should be fitted with an interrupting handle.

A suitable *galvanic battery* is more expensive, for it requires at least 24 small Leclanché cells (capable of giving about 35 to 40 volts), a collecting board and reverser, with electrodes as before, and, to be complete, a galvanometer that has been properly tested (Fig. 156). The constant current can be used from the main with a suitable switchboard.

To *test the faradic reaction* place the large electrode in the patient's hand, on the back of the neck or some other indifferent position, and another electrode connected with the interrupting handle over the motor point of the nerve or muscle to be tested. A knowledge of the motor points of nerve and muscles is not indispensable, for a great deal may be learned by rubbing the medium-sized electrode with plenty of water well over the limb. The motor point of a muscle is near the point of entry of its nerve; that of a nerve is generally near its most superficial part. The electrodes and the skin should be very *thoroughly* wetted with plain warm water. Some prefer salt and water, but this damages the instruments more and does not increase the conductivity, provided the electrodes are kept clean with a little soap occasionally. The patient should be so placed in a good light that both sides of the body can be seen equally. Having thoroughly *wetted the skin on the two sides* you are about to compare, ascertain first (by gradually sliding up the secondary coil) what is the minimum current necessary to produce a minimum contraction of the muscle or muscles on the healthy side. Then, secondly, test the side suspected of disease with the same amount of current, to see if the same degree of contraction is produced, or if not, what strength of current is requisite.

The faradic contraction of a muscle can only be obtained through its nerve, *i.e.*, when a healthy nerve is connected with it, however healthy or unhealthy the muscle itself may be, and therefore the faradic current is an accurate test for the condition of the nerve. If the nerve is damaged or destroyed there will be a proportionate diminution or total loss of the faradic muscular reaction. This fact was discovered in 1859 by Baierlacher, who experimented on the facial nerve. *A diminution of faradic contraction signifies a lesion of some part of the lower motor neuron.*

To *test the galvanic reaction* the electrodes are placed in the same position, and for (a) *quantitative* alterations, compare the two sides as before, noting what amount of current, as indicated by the number of cells used or the number of m.a. registered by the galvanometer, is required to produce a minimal contraction on both sides when both sides have been wetted equally. (b) The *qualitative* alterations are a trifle more intricate. Begin with the kathode (negative pole) placed on or about the motor point under investigation. Close the current by means of the interrupting handle, and the contraction obtained is known as the Kathodal Closing Contraction (K.C.C.). The kathode or negative pole is the one connected with the zinc element in the cell. If you cannot tell which is the kathode, place the two wires in a glass of water, and a lively production of gas comes off at the kathode. Next convert the electrode on the patient into the anode or positive pole by means of your reverser, and repeat the process of closing the current. The resulting contraction is called the Anodal Closing Contraction (A.C.C.). These are the two important data to obtain, for normally with the same strength of current and the same degree of wetting of the skin K.C.C. is greater than A.C.C. (K.C.C.>A.C.C.), or what amounts to the same thing, a greater strength of current is required to produce A.C.C. than K.C.C.

Muscular contraction with a galvanic current is only produced at its closing or opening. Sometimes the opening contractions are tested, the normal order being K.C.C.>A.C.C.>A.O.C.>K.O.C.

Abnormally A.C.C. is equal to or greater than K.C.C.

The *reaction of degeneration* (R.D.) differs in different stages. When a nerve is severed or is the seat of acute inflammation, after a preliminary increased electrical reaction to both currents during the first two days, (a) the reaction is gradually lost to both currents *during the ensuing ten days*, the faradic reaction not being regained unless regeneration takes place.

(b) In the *second or third week* and for some weeks afterwards the galvanic reaction is restored and Erb's reaction of degeneration is met with in its typical form, and consists of—

(i.) no muscular contraction to faradism, however strong the current;
(ii.) a quantitative increased contraction to the galvanic current in all forms;
(iii.) the galvanic contraction which in health is prompt and sharp becomes sluggish;
(iv.) qualitative galvanic changes are present in that A.C.C. is equal to or greater than K.C.C.

(c) *Two or three months later* the galvanic contractility gradually disappears (unless regeneration is established), though for one or two years A.C.C. can be obtained with a gradually increasing strength of current.

The **diagnostic** value of electricity is very great, for the R.D. is present in greater or less degree in all severe diseases affecting the anterior horns, anterior roots or peripheral nerves (motor or mixed); in short, any part of the lower motor neuron; while it is absent in diseases of the upper motor neuron.

List of diseases in which the Reaction of Degeneration is found :—

I. Diseases of the *anterior horns.*
  1. Acute anterior poliomyelitis.
  2. Sub-acute and chronic anterior poliomyelitis (*e.g.*, spinal form of progressive muscular atrophy).
  3. Amyotrophic lateral sclerosis.
  4. Syringomyelia and spinal gliosis.
  5. Cervical myelitis, transverse lumbosacral myelitis, in the arms or legs respectively.
  6. Diseases of the motor nuclei in the medulla corresponding to the anterior horns (progressive bulbar paralysis, acute inferior polio-encephalitis).

     In chronic anterior poliomyelitis (progressive muscular atrophy) there is a slow progressive involvement of the multipolar cells, and, therefore, irregular electrical results are obtained, because one part of a muscle may be more involved than another. In idiopathic myopathy irregular results are also obtained.

II. Diseases of the *anterior roots.*
  1. Compression from tumours and thickened membranes (*e.g.*, in syphilis, and hypertrophic cervical pachymeningitis).
  2. Compression in the intervertebral foramina in vertebral diseases (caries, tumours, fracture, luxation).

III. Severe diseases of the *peripheral nerves.*
  1. Injury or pressure from tumours, bruises, operations, etc.
  2. Rheumatic (*e.g.*, in facial paralysis), and other toxic and infectious causes—*e.g.*, lead, alcohol, arsenic, diphtheria, and other infections.

In regard to **prognosis**, whenever faradic reaction is retained, even if only slight with a very strong current, restoration is possible; if it is quite lost for a few weeks restoration is still possible; but if it remains totally lost after several months there is but little prospect. Whenever an incomplete R.D. is present the prospect of recovery may be considered even after notable atrophy. Slight voluntary movement sometimes returns *before* the return of the electrical reactions.

The slighter the lesion the less the alteration in electrical excitability. Slight compression of a nerve enough to produce paralysis may produce but little alteration, though in nearly all cases the faradic excitability is somewhat diminished.

PART C. DISEASES OF THE NERVOUS SYSTEM. THEIR DIAGNOSIS, PROGNOSIS AND TREATMENT.

§ 517. Routine Procedure and Classification.

First, as on previous occasions, we ascertain and verify the patient's LEADING SYMPTOM, which will probably be one of those mentioned in Parts A. or B.

Secondly, interrogate the patient as to the HISTORY OF THE ILLNESS, his PREVIOUS, and FAMILY HISTORIES, all of which require special care in nervous diseases (as mentioned in § 511).

Thirdly, the PHYSICAL EXAMINATION has been dealt with systematically in Part B., but for a **routine examination** in ordinary cases it is convenient to adopt a regional method, beginning at the head and proceeding downwards.

a. The HEAD. Enquire as to intelligence, sleep, pain, "attacks" or head sensations of any kind.

Eyes—vision, abnormality of pupils, squint, ptosis, nystagmus; and later, ophthalmoscopic examination.

Face—any defect of speech, tremor of the lips, or immobility

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Test the muscles by such directions as "show me your teeth"; "screw up your eyes"; "put out your tongue."

Hearing and other *cranial nerves*, as may be necessary, in numerical order.

b. Upper limbs. Examine the state of the muscles, and compare the force of the grasp of the two sides.

Elicit any *tremor* or involuntary movement by extending the hands and fingers; and direct the patient to touch the tip of his nose with his forefinger, repeating the performance with eyes closed (ataxy).

Test the supinator and triceps *reflexes.*

c. Lower limbs. Examine the muscles for paralysis, rigidity, flaccidity, or wasting.

Walking—Note any peculiarity in the pose or the *gait;* can the patient stand with heels together and eyes closed (Romberg's test).

Examine the *knee-jerks,* test for *ankle-clonus.*

d. Test for abnormalities of common sensation (touch, pain, and temperature).

Enquire as to the state of the sphincters.

Remember that there are two steps or stages in the diagnosis of diseases of the nervous system, which, in order, are—1st, **localisation**, namely, what part of the nervous system is the seat of disease? 2ndly, what is the **nature** of the disease in that locality?

If the symptoms point to some **generalised neurosis** turn first to Group I. (below).	
If to defect of **consciousness** or the **mind** (Group II.)	§ 524
If there is definite **pyrexia** (Group III.)	§ 540
If the symptoms relate to the **muscular system** (Group IV.)—	
Paralysis	§ 545
Inco-ordination or defect of gait	§ 571
Rigidity	§ 576
Tremor	§ 583
Convulsions	§ 591
Muscular atrophy	§ 594
If to some **sensory** or **painful** disorder (Group V.)	§ 598
If to the **special senses** or cranial nerves (Group VI.)	§ 602
If there is some deformity of the **skull** (Group VII.)	§ 620

GROUP I. GENERALISED NEUROSES.

The word neurosis connotes a functional disorder of the nervous system; and by generalised neuroses are meant those which present generalised symptoms. The members of this group have several interesting features in common; they present no physical signs during life; after death no definite lesions have been discovered by the means yet at our disposal (on which account they are known as functional disorders); they are rarely or never fatal of themselves; and they run a prolonged but variable course and present a proneness to paroxysmal exacerbations, which in some amount to attacks or "storms." The remarkable resemblance of chronic alcoholism, morphinism and toxic neurasthenia in their clinical features to the other members of this group, suggests that all the cases here met with may be dependent on some disorder of the blood.

I. Neurasthenia.
II. Hysteria.
III. Alcoholism.
IV. Morphinism and other drug habits.
V. Hypochondriasis.
VI. Collapse.

§ 518. **Neurasthenia** is an irritable weakness of the nervous system which may arise from a great variety of causes, and may result in many and various symptoms of nervous, mental, and bodily inefficiency.

The *symptoms* are of a subjective order. The patient may come for many different reasons—(1) a feeling of "weakness and nervousness" is one of the most usual complaints, or he may state that he is easily tired, easily startled, easily upset. The physical debility or disability is sometimes less marked than the mental, but it is often severe and may be sufficient to confine the patient to bed. Slight anaemia and loss of weight may also be present. There are no physical signs unless the occasional presence of exaggerated knee-jerks, retraction of the fields of vision, or dilatation of the pupils may be so considered. It is convenient to describe a cerebral, cerebro-spinal and spinal type according to the prevailing symptoms. (2) Cerebral or mental symptoms are always present and generally predominate. Everything the patient has to do is a trouble and a source of worry, and sometimes the simplest mental work such as adding up a column of figures is impossible. The sleep is disturbed by dreams, or there is insomnia, or "startings" in the sleep. All the special senses are easily tired, and sometimes the patient is quite unable to read. The pupils are usually dilated and reaction to light and accommodation deficient. The memory and the power of concentrating the attention are defective. Some patients are *irritable*, egotistical and exacting; others *gloomy* and melancholic, keeping their troubles to themselves. Anxiety and morbid dreads form a prominent feature, sometimes indefinite, the fear of some unknown evil, sometimes definite such as agarophobia (fright when being in an open space), claustrophobia (fear of going into churches or other buildings), monophobia (fear of being alone), antheropophobia (dread of society or of people). Sometimes the dreads take the form of paroxysmal panics or terrors which pass off as suddenly as they come, and I have seen several of such cases pass on to melancholia and dementia.[1] There is no doubt that the miseries they suffer are very real, and not a few patients, particularly cases of the gloomy type—those who go wearily on with their work saying but little of what they feel, who have probably never threatened suicide—seek relief by terminating their lives. Other patients drift into a chronic and incurable hypochondriasis. (3) Spinal and musculo-sensory symptoms are also present in greater or less degree—restlessness and jerking of the limbs, weariness on the least exertion, vague pains in the back and limbs, generalised tenderness or a hypersensitive condition of the whole body, neuralgic pains and tender spots. There may be fine muscular tremor; but never localised paralysis or hemianæsthesia as in hysteria, and never absent knee-jerks as in tabes dorsalis. Absence of knee-jerks excludes the diagnosis of neurasthenia. Spermatorrhœa, nocturnal emissions, or the discharge of glairy fluid at stool, sexual disability, rarely increase, and still more rarely perversion, are met with. (4) There are generally symptoms which may be aptly attributed to disturbance of the vaso-motor sympathetic system, *e.g.*, long-drawn sighs, causeless palpitation, attacks of flushing followed by shivering, a sensation of "pins and needles" in the limbs, cold hands and feet. Palpitation of the heart and various "throbbings," giddiness, and faint feelings are also met with. It is in the presence of these sympathetic symptoms and attacks that neurasthenia overlaps hysteria. (5) According to some authors gastric symptoms form an essential part of the symptoms of neurasthenia, and this is often but not always true. In a great many cases gastro-intestinal disorder is the cause and accompaniment of neurasthenia, and in others the asthenia of the nervous system has given rise to gastric atony or, as they say, gastric neurasthenia (see etiology).

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[1] "Clinical Lectures on Neurasthenia," 2nd edition, J. H. Glaisher & Co., London, 1902.

TABLE OF DIAGNOSIS.

	Neurasthenia.	Hysteria.	Hypochondriasis.
Sex.	Both sexes almost equally.	Female sex almost exclusively.	Males chiefly affected.
Age.	Any age may be affected.	Definite manifestations of some kind appear for the first time practically always before 25, generally between 15 and 20.	Rare under 30; predisposition from 30 to 50.
Causes.	Produced by overwork; gastro-intestinal and other causes of malnutrition, or defective metabolism: occasionally by shock.	Subjects of the hysterical diathesis are liable to hysterical attacks or symptoms *throughout life.* The determining cause of active hysterical manifestations always an emotional upset.	Solitary, sedentary life; prolonged gastro-intestinal troubles.
Onset.	Starts gradually and runs a fairly even course.	Onset sudden, generally with an attack of some kind; all phenomena vary from hour to hour and day to day.	Starts gradually, and runs an even and intractable course of indefinite duration.
Mind.	Mental exhaustion and inability to think, study, or do work; memory deficient; intellect clouded for business; always tired; temper irritable; depression rather than sadness; not equal to the exertion of amusement.	Wayward, impulsive, and emotional; fond of gaiety and amusement; usually joyous, but laughter and tears alternate with great rapidity; memory and intellect sometimes brilliant, rarely deficient.	Introspective habit; observing accessible organs and secretions; habitual sadness; no taste for amusement; patient tries an endless succession of remedies and doctors; always striving for a cure.
Symptoms.	Chronic weakness and nervousness; rarely attacks of vague sensations about the head; convulsions never; gastro-intestinal trouble of some kind in 91 per cent.	Symptoms paroxysmal; seizures of different kinds frequent; flush readily; attacks of globus and syncope frequent; convulsive attacks in nearly half the cases.	No seizures of any kind; runs an even course.
Result.	May last a long time, but by appropriate measures it is gradually CURABLE.	Active manifestations disappear suddenly and unexpectedly; very apt to recur, and therefore only SEMI-CURABLE.	Once established hypochondriasis is impossible to eradicate: usually progressive and INCURABLE.

In the *diagnosis* the very vagueness of the symptoms is an aid. Neurasthenia is certainly not the same disease as *hysteria* (see table), nor is neurasthenia a new name for hysteria, as some suppose, though they overlap in some respects. Hysterical phenomena are paroxysmal and recurrent, with intervals of health, throughout life ; in neurasthenia the symptoms are more or less continuous, occur almost equally in either sex, and are liable to come on at any age.

The diagnosis from early *tabes* and *General Paralysis of the Insane* is sometimes extremely difficult. In neurasthenia the knee-jerks are never, in my experience, absent, and the typical Argyll-Robertson pupil never present. For the diagnosis of G. P. I. chief reliance has to be placed on the typical articulation and the tremor of the lips and tongue.

Prognosis. Neurasthenia is essentially a chronic disease and leads to a great deal of misery, but is never fatal, excepting by suicide. Some hold that neurasthenia is incurable. The author, however, believes it to be mostly curable, provided a careful investigation be made, leading to the discovery of the cause or causes in operation. It may run on for years, especially if, as is so frequently the case, dyspepsia, colitis, or some other chronic cause is in operation. Its prognosis depends chiefly on (i.) its previous duration, and (ii.) the removability of the cause, and (iii.) the age of the patient, being more favourable in younger persons.

Among the *causes*, undoubtedly long neglected dyspepsia, bolting of food, colitis, or some other abdominal trouble is by far the most frequent in my experience. My statistics (*loc. cit.*) show that some underlying gastro-intestinal cause is present in 91 per cent. of the cases. An hereditary predisposition plays a part in the etiology—though not in my belief as prominent a one as some hold—either by reason of a neuropathic predisposition in the progenitors, or alcoholism or tuberculosis and other debilitating agencies in the parents. In regard to age none are exempt, but neurasthenia is commoner in young and middle-aged adults than in advanced age or in children ; and 61 per cent. of the cases under my care were males. A sedentary indoor life in town appears to predispose, and the rush and strain of modern civilisation favours the occurrence of the disease. It has been my practice to classify the causes of neurasthenia into four groups, compound causes being frequently in operation:—(1) *Toxæmic* causes which include dyspepsia, colitis, and other gastro-intestinal disorders such as chronic constipation, bad teeth or pyorrhœa alveolaris. Abuse of alcohol, morphia or cocaine, and Graves' disease, may also be included here. (2) *Malnutrition* causes—such as post-influenzal (a specially potent cause) and other post-febrile and debilitating conditions, and deficient or defective food. (3) *Fatigue* and over-functioning are factors which include prolonged overwork, worry, anxiety, deficient or defective sleep, severe prolonged pain and excessive venery. (4) *Emotional* and *traumatic* causes—grief, shock (mental or bodily), railway and other accidents, in which the neurasthenia need not supervene until a few weeks after the shock. Injury may produce either "traumatic neurasthenia" or "railway spine" (see paraplegia). In addition to these four causes, I have met with various *reflex causes*—floating kidney and other enteroptosis, eye-strain, cardiac, pulmonary and any other local diseases.

The *treatment* resolves itself briefly into—(1) The alleviation of the distressing symptoms as far as may be. Bromides are particularly useful in this respect; morphia and other sedatives require the greatest care. Alcohol and tobacco should be avoided. (2) The removal of or compensation for the above causes. Dyspepsia is in evidence in by far the largest number of neurasthenics in out-patient work, and it is surprising how efficacious an alkaline gentian mixture is in the majority of cases. Aperients and intestinal antiseptics are valuable. Regular outdoor exercise is useful. (3) Nerve tonics with hygienic and educational measures conducted with sympathy and encouragement will do much for those in whom hereditary predisposition is strong. Among nerve tonics strychnine is very useful, and arsenic, phosphorus, damiana, phosphates, glycerophosphates, malt, and cod-liver oil may be tried. Turkish and warm baths allay irritability. Cold baths and judicious hydrotherapy, various forms of electricity and a regulated diet have all done service in these cases. (4) Complete physiological rest of the nervous system is of the greatest value, but I cannot recommend Weir-Mitchell treatment, at least in its complete form. Sea voyages are highly beneficial.

§ 519. **Hysteria** is a word which is applied in two senses: sometimes it is used for the *diathesis* or tendency in the individual to the development of hysterical manifestations, and sometimes it is applied to those *manifestations* or disorders themselves. In the former sense hysteria may be defined as a condition of instability of all the emotional, vaso-motor, and sympathetic reflex functions, with a tendency to the development throughout life of many different forms of nervous seizure, and of various motor and sensory disorders closely resembling organic diseases of the nervous system, seldom or never leading to a fatal issue.

Symptoms of the HYSTERICAL DIATHESIS. There is no particular form of facies uniformly attaching to this condition; but the hysterical disposition is essentially one of unstable equilibrium of the emotional and other faculties of the mind. These patients are easily aroused to violent expressions of feeling, hasty judgments, impulsive actions, and to passionate exhibitions of various kinds. There is a hypersensitiveness to all forms of pain, and a tendency to neuralgia, a favourite seat for which is just below the left breast, or on one side of the head. The hysterical diathesis in an individual may also be suspected by the presence or a history of any of the 5 following symptoms, which may with propriety be called the "hysterical stigmata."[1] (1) *Flushings* of the face and other parts with or without provocation is probably the most frequent. Sudden pallor and other vaso-motor skin phenomena[2] are also very frequent in hysterical subjects. (2) *Nervous attacks* of some kind are sure to occur in hysterical subjects sooner or later—"hysterics" (crying and laughing), nervous faints, etc. They are specially apt to occur at the catamenial period or after some slight emotional shock. (3) "*Globus*" is a sensation as of a ball in the throat, or a sense of choking or suffocation. Flatulence is a frequent accompaniment of globus, and a severe attack of globus with prostration is often terminated by copious windy eructations. (4) "*Ovarie*" or the hysterical ovarian phenomenon consists of a tenderness on pressure on either inguinal region, which produces an indescribable feeling rising up towards the heart and throat. Pressure in this, the "ovarian" region, may determine some kind of attack. This phenomenon is in no way dependent on the ovary, but as I have elsewhere shown,[1] is specially related to the ilio-hypogastric and ilio-inguinal nerves. Similar hyperæsthetic or hysterogenic zones may exist elsewhere. (5) *Patches of anæsthesia or hyperæsthesia* may exist almost unknown to the patient. The anæsthesia may occupy one half of the body and involve the special senses on that side.

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[1] A stigma etymologically signifies a paramount or abiding mark or sign by which something may be recognised.

[2] See a clinical lecture on the Skin Symptoms of Hysteria, *The Lancet*, Jan. 30, 1904.

Hysterical Disorders may affect any part of the body, closely simulating organic disease. These are described under their suitable sections, and a brief summary only is given here. (1) Disorders of motion—paralysis of the voluntary muscles, hemiplegia, monoplegia, paraplegia, rarely if ever paralysis of the face; tremors and convulsions, tonic and clonic spasm of one or several limbs, or involving the whole body. (2) The involuntary muscles may also be affected by paralysis or spasm—adductor spasm or paralysis of the vocal cords, dysphagia, hiccough, cough, dyspnœa, borborygmi, phantom tumour, vomiting without nausea. (3) Anæsthesia may affect one limb or half of the body; hyperæsthesia, especially of the spine, and various neuralgiæ are common. (4) Joint affections with pain and swelling may occur. (5) Hyperpyrexia is said by some to occur with or without local manifestations of hysteria. (6) The special senses may be affected—amaurosis, hemianopsia, retraction of the field of vision, deafness, disturbance of taste and smell. (7) Finally there may be mental changes—trance, catalepsy, hallucinations and delusions, especially after convulsive attacks.[2]

The *diagnosis* of hysteria from neurasthenia has been considered in § 518. The salient features of hysteria are its limitation to the female sex and the *paroxysmal occurrence* of all its symptoms. The diagnosis of the numerous hysterical phenomena will be dealt with under the various disorders which they most resemble.

Prognosis. The hysterical diathesis lasts throughout the lifetime of an individual, modified from time to time by surrounding circumstances—once hysterical always liable to develop hysterical manifestations. The disease never terminates fatally of itself, but it often renders the life of an individual a misery to herself and those around her.

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[1] *The Lancet*, July 20, 1901, p. 122.

[2] The relative frequency of the various manifestations of hysteria is given in "Lectures on the Pathology of Hysteria," *Clinical Journal*, May 25 and June 1, 1904; *The Lancet*, Jan. 30, 1904.

Etiology. Hysteria is practically confined to the female sex; it occasionally presents itself in the male sex, but only to the extent of about 0·2 per cent. Heredity is a potent factor, and can be traced in at least 75 or 80 per cent.; the influence is transmitted particularly through the mother. In other cases there is a family history of one or other of the diseases dealt with in this group (Group I.). Faulty education or a life of self-indulgence, or any mode of existence which leads to introspection, a diminution or abolition of the control normally exercised by the will, undoubtedly foster the evolution of the diathesis. Faulty hygienic and physical development in childhood also favour the occurrence of hysterical phenomena in after life. The favourite ages when evidences of the hysterical diathesis are most manifest, and when the various phenomena are most likely to supervene are soon after the evolution and at the involution of a woman's sexual life. The determining cause of all hysterical manifestations is some emotional shock, trivial or severe. No anatomical or histological lesions have yet been discovered.

Treatment. Educational treatment is very important in these cases, and as there are but few mothers possessing the combination of judgment, firmness, tact and kindness which is necessary in the treatment of their own daughters, subjects of hysteria should be entrusted to others. Some regular occupation and interest in life is another means by which the temperament may become eradicated. Nothing to do or a frivolous kind of existence is calculated to foster and develop the diathesis. Matrimony thus becomes a valuable adjuvant, because it gives to a young woman occupation, interests, and responsibilities outside herself. Briquet[1] showed conclusively that hysteria had no causal relation to ungratified sexual passions. If the manifestations of the diathesis are sufficiently pronounced, a course of treatment is indicated which comprises (1) removal from the conditions under which the disease is fostered, (2) isolation from sympathetic friends, combined with (3) over-feeding, a large portion of the diet consisting of milk and other easily assimilable foods. (4) Massage is undoubtedly a great help, as enabling the patient to take and assimilate more food. These 4 measures constitute the Charcot or Weir-Mitchell method of treatment. The treatment for the nervous attacks, when they arise, consists of the sudden application of cold water to the face, the faradic battery to the limbs, and the internal administration of asafœtida, valerian, and spirits of ether, or chloroform.

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[1] "Traité Clinique et Thérapeutique de l'Hystérie," par le Docteur Paul Briquet, p. 206. Paris, Baillière et Fils, 1859.

§ **520. Alcoholism** or excessive indulgence in alcohol is met with clinically in 3 forms—(1) acute alcoholism, (2) chronic alcoholism, a phase of which constitutes (3) delirium tremens.

Acute Alcoholism is due to an excessive quantity taken in a few hours; it gives rise to mental disturbance, muscular incoordination, and finally narcosis with marked lowering of the body temperature, and a heavy alcoholic odour of the breath. The stupor of apoplexy, uræmia and other causes of coma (§ 525), and the muttering delirium (§ 346) which occurs in pneumonia and other diseases in alcoholic subjects are apt to be mistaken for drunkenness, a serious error which is best avoided by keeping a patient under observation and suspending our judgment.

Chronic Alcoholism is due to the persistent imbibition of moderate doses of alcohol over a long period of time. The effects are worse when taken on an empty stomach or in the form of raw spirits. It acts as a tissue poison on the nervous, muscular (voluntary and involuntary), and epithelial elements; and hinders tissue oxidation, so leading to fatty degeneration.

The *consequences and symptoms* in the earlier phases resemble neurasthenia in many respects, with special toxic effects added later. The *neuro-muscular system* early shows signs in (i.) the unsteadiness and tremor of muscles, especially those of the hands, and peripheral neuritis, of which alcohol is the most frequent cause. (ii.) The mental processes become slow, the memory, judgment, and will enfeebled, and later dementia or some other form of insanity ensues. Delirium tremens (below) supervenes from time to time and sometimes epileptiform convulsions. The *digestive* *system* is quite as commonly affected. (i.) Chronic gastric catarrh attended by characteristic morning vomiting is always present in spirit drinkers, and gastric dilatation in beer drinkers. (ii.) Hepatic congestion occurs in all cases, and cirrhosis, with or without fatty degeneration, in a good number, but not in all. The *heart* dilates and undergoes fatty degeneration, and the vessels become thick and degenerated. The *kidneys* become congested, enlarged and later cirrhotic. The *facies* of the chronic toper is characteristic—redness of the cheeks and nose with œdema of the conjunctivæ.

Outbreaks of **delirium tremens** (delirium e potu) supervene from time to time in the course of chronic alcoholism ; it never occurs as the result of one orgie in a usually temperate person. It is determined sometimes by an unusual drinking bout, sometimes by cutting off the habitual libations, sometimes as the result of an injury or the advent of pneumonia or some other inflammatory affection. Incoherent mutterings or ravings characterised by hallucinations of vision (reptiles, rats or monsters) accompanied by muscular tremor, intractable sleeplessness, and in bad cases 2 or 3 degrees of fever, are the leading features of the malady ; which usually runs its course in 2 to 5 days.

The *diagnosis* of chronic alcoholism is generally easy. The diagnosis of delirium tremens is referred to on p. 576. Care should be taken not to overlook acute pneumonia, particularly of the apex. The *prognosis* of delirium tremens for recovery is generally favourable if the temperature is not much elevated and the strength of the patient can be maintained.

The *treatment* of acute alcoholism consists of the administration of an emetic such as zinc sulphate, or apomorphine $\frac{1}{6}$th of a grain hypodermically. (2) A chronic alcoholic habit is rarely abandoned after 40, and residence in a home is advisable in all confirmed cases, to enable the patient to regain his self-control. In other cases much may be done by careful domestic control and medical supervision. The gastric catarrh must be treated, and the uncomfortable "sinking" feelings may be much relieved by tincture of capsicum, peppermint or chloroform water. In acute and chronic alcoholism one of the most distressing symptoms is insomnia, and it is difficult to treat. In the acute form opium is said to be contra-indicated, and it is doubtful if chloral or other hypnotics will shorten the attack. Digitalis in large doses may be tried, but it will be found that large doses of bromides and in some cases opium are generally successful. For dipsomaniacs (who have periodic outbreaks) and those who are born with an enfeebled nervous system, little can be done unless they will surrender the control of their lives to others. The great value of strychnine, atropine and cinchona in the treatment of alcoholism and morphinism has no doubt been known to several observers for some years, and I cannot speak too highly of them as regards my own experience. Some years ago a "gold cure" was "invented," and advertised,[1] but the active agents of this cure were undoubtedly the remedies above named, not the auric chloride. Strychnine and atropine sulphates (hypodermically) gr. $\frac{1}{60}$th and $\frac{1}{120}$th respectively may be given with cinchona bark (by the mouth) 4 times daily until the throat is dry and the pupils dilated.[2]

The treatment of *delirium tremens* calls in the first place for a reliable attendant (perhaps two or three), for the patient may be violent in his attempts to escape from his horrible visions, and artificial restraint may be necessary. To procure sleep and maintain the strength by nourishment are the main indications. Large doses of bromide, and chloral (if the pulse is not too weak) or hyoscine gr. $\frac{1}{100}$ may be tried; but as a rule the malady runs its course unaffected by drugs. Graduated cold baths (§ 393) and cold packs are often very efficacious in cases with pyrexia.

§ 521. **Morphinism** (syn. morphia habit, morphinomania) and other **drug habits.** Hypodermically, **morphia** in small doses is a nerve stimulant as well as a hypnotic, and induces a feeling of contentment and well-being. But in the course of 24 hours reaction and craving for more occur, particularly when pain is present, and by degrees the dose has to be increased until in the course of a few months 20 to 100 times the normal dose is necessary to produce a feeling of satisfaction, and can be easily tolerated. If such a patient is suddenly deprived of the drug, the following *symptoms* (which I have been accustomed to call

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[1] The following is the analysis, supplied to me by Mr. Wootton, of Luton, Beds, of the "gold cure" in question—

The *injection*, 4 times daily. Strych. sulph. gr. $\frac{1}{2}$; atropin sulph. gr. $\frac{1}{4}$: ac. boric. gr. 15; aq. ℥iv.

The *mixture*, ʒj. every 2 hours when awake. Ammon. hydrochlor. gr. j.; aloin, gr. ij.; tr. cinch. co. ℥iij.; aq. dest. (better sp. chlor.) ℥j. Morph. used at outset of treatment also.

Red Mixture for alcoholics. ʒj. every 2 hours when awake. Sodio-auric chloride, gr. xij.; amm. chloride, gr. vj.; strych. nitrate, gr. j.; atropin, gr. $\frac{1}{4}$; ext. cinch. liq. ℥iij.; ext. cocæ liq. ʒj.; glycerin, ℥j.; aquæ, ℥j.

[2] Mr. S. B. Fenn, *Brit. Med. Journ.*, 1904, vol. i., p. 1008; and Dr. C. A. McBride. *Brit. Med. Journ.*, 1904, vol. i., p. 1006.

"amorphinism") set in. The pulse, which was previously normal, becomes rapid and of low tension, the arteries dilated, and the patient prostrate, suffering agonies from the tingling in the limbs, sweatings, sneezing, lachrymation, diarrhœa, vomiting, uncontrollable restlessness, faintings, sinkings in the pit of the stomach, extreme wakefulness, and a host of horrible and indescribable somatic sensations resembling extreme neurasthenia. The only *signs* by which the *morphine habitués* can be detected are contracted pupils, pallor of the face, the frequency with which they withdraw to satisfy their craving, a difference being observed in their depression before and their gaiety and brightness afterwards.

Consequences of the morphia habit. Enormous doses may be taken by gradual increase; one of the largest in my experience was 25 grs. a day, reached after a habit of only two years. At first the patient is always gay, and has great capacity for mental and bodily endurance. But if the habit be continued the character gradually becomes wholly altered, and various moral obliquities become manifest. The patient alienates his friends by his tempers and unreliability; and one by one truth, reverence, virtue, and honesty disappear. If there be difficulty in procuring the drug, great craftiness is exhibited, and cases are known of women previously of the highest character selling their virtue, and their husband's honour, to procure it. In course of time the mental powers gradually degenerate, and suicide is not infrequent in those who desire but are unable to rid themselves of the thraldom. The body also suffers, and the patients become pale, and generally emaciated. They get careless in the use of their syringe, multiple abscesses form and death may result from septicæmia. Fatty degeneration of the viscera ensues, especially of the heart, as in the case of a girl aged 19 which was reported in Paris some years ago. My own belief is that many of the cases of sudden death reported as due to overdose of morphia might be shown to be due to this condition.

Prognosis. That the habit shortens life is certain, though it may go on for many years. The danger of sudden death, due to causes just explained, or from an overdose, is also considerable. The curability of a case of morphinism depends in my experience on three points; the age of the patient, the duration of the habit, and the curability of the painful affection for which it was first contracted. The actual quantity per diem which has been reached is of small account. A habit of 4 grains daily of 10 years' duration was more difficult to cure than one of 22 grains of 3 years' duration. If carcinoma or some other cause of an incurable and recurrent pain be present, and especially if the patient is aged, the tendency to relapse is great, and it may be impossible to ease the pain in any other way.

Treatment. (a) To break the habit the patient must place himself under the *absolute control* and guidance of a physician in whom he has confidence. Three methods are advocated—sudden cessation, gradual reduction, and gradual reduction combined with the substitution of solid opium or morphia by mouth or rectum. The first of these should never be employed unless time is an object, for the suffering is very great, and if the habit be of long duration, not without danger. In the third, which is advocated by Dr. Oscar Jennings of Paris, a definite scheme should be written out by the physician, in which double the quantity of opium, by mouth or rectum, replaces the reduction in the morphia. Afterwards when all hypodermics have ceased the opium internally can be gradually reduced without much difficulty. The second method, gradual reduction, is the one which I have found most successful. There should be no difficulty in reducing, by ¼ grain a day, down to 2 grains, after which the reduction should continue in quantities less and less in a geometrical ratio. (b) Treatment of the symptoms of amorphinism. The prostration and somatic sensations may be partially relieved by moderate doses of alcohol, ammonia, and other stimulants, or by strychnine ♏iv. hypodermically and atropine, as recommended on p. 928. Digitalis and other drugs which aid the heart and contract the vessels are useful. For the vomiting and diarrhœa, bismuth is best, but the diarrhœa should not be checked too much. Dionine in one case under my observation relieved the restlessness and craving. For the sleeplessness, chloral and other hypnotics and analgesics may be tried, but none are of great use. After recovery there are two tendencies—(1) towards relapse, and therefore the patient should be kept under observation, and (2) towards alcoholism, therefore great care is required in the administration of stimulants.

The **cocaine habit** leads to many of the troubles of the morphia habit, only there is a greater tendency to mental symptoms and mania. Morphia and cocaine is a frequent combination and in such case the cocaine may, with comparative ease, be first withdrawn. Then the morphia reduction may be proceeded with as above described.

The **chloral habit** is not so common nowadays as the preceding. The consequences or symptoms consist of gastro-intestinal disturbance, lowered nutrition, pains, skin eruptions, depression and irritability. Sudden death may occur from slight increase of the dose.

Sulphonal, phenacetin, antipyrin and other tar products do not so readily engender a craving, but when habitually used the patient cannot do without them, and in course of time symptoms as in chloral hydrate arise.

§ 522. **Hypochondriasis** is a morbid condition of the nervous system allied to neurasthenia on the one hand and melancholia on the other. It is an introspective, melancholic, or pessimistic habit of the mind, in which the patient believes, without cause, that he is the subject of one or more serious bodily disorders. He is gloomy, reserved, and wrapped up in himself; a slight pain in the stomach is certainly cancer; a slight palpitation is regarded as mortal cardiac disorder; or the testicles hang too low, and therefore he will certainly become impotent for life. Yet, in spite of all he is not without hope, for he will spend his life taking physic, and frequenting the consulting rooms of physicians, surgeons, and quacks. Nor is he suicidal; two points in which the condition differs from true melancholia, which is a state of hopelessness and a tendency to self-destruction.

Diagnosis. Hypochondriasis used to be regarded as the representative of *hysteria* in the male sex; but it is in reality a very different malady (table in § 518). In the former one does not meet with the "attacks," paralysis, and sensory alterations so frequent in hysteria. Hypochondriasis bears a closer relation to *neurasthenia*, but the latter is a definite disease, having its own symptoms.

Causes. Hypochondriasis is occasionally seen in the female, about the menopause, but the patients are mostly men of middle age. It is rarely seen before puberty, or indeed before 30, and generally makes its first appearance between 30 and 40. There is often a neurotic family history, and often of insanity. Dyspepsia or intestinal disorder is always present, and may be looked upon as its most frequent cause, a fact which is interesting when we remember the marked prostration and depression of the vital powers which occur in all acute abdominal diseases; and the depression of spirits, with irritability of temper which attend all stomach and liver disorders. Flatulence is a common symptom, and the stomach is often dilated. Dispersion of this wind in the stomach gives marked relief; another fact pointing to the abdominal sympathetic as being concerned in the disease.

Treatment is neither easy nor satisfactory. The dyspepsia should be treated, and a draught of ammoniated tincture of valerian, or pil. asaf. co., or some other anti-spasmodic taken occasionally for the flatulence. It gives relief, not only to the flatulence but to other symptoms. The bowels should be carefully regulated. These means, with regular exercise, constant change, cheerful society, help to break through the vicious attitude of the mind; but complete recovery is very rare.

§ 523. **Collapse** (or **shock**) is an acute condition of extreme bodily weakness with exhaustion of the nervous system, an extreme state of prostration. An attempt is sometimes made to distinguish shock and collapse, but the two are clinically identical. The term shock is applied to that condition which follows any sudden mental or physical injury; the term collapse when it supervenes on some less sudden cause, such as cholera. The condition in either case is a serious one. The pathology is still obscure, but in effect there is paralysis, or more properly paresis, of all the muscular tissues of the body, voluntary and involuntary (muscles of the limbs, of respiration, of the heart and arteries). The *symptoms* may be arranged under the following headings:—(1) the skin is pale, cold and clammy (especially of the extremities): the surface temperature is 2° or more under normal; the pupils are dilated and react slowly to light. (2) The circulation and respiration are very feeble, the pulse being rapid and scarcely perceptible. (3) Loss of voluntary movement (sometimes restlessness and, in cases of profuse hæmorrhage, convulsions). The mind is apathetic, but the intellect is clear. The urine and other secretions are diminished or suppressed. The patient may die, or may pass into a reaction stage with slight pyrexia.

Diagnosis. In *coma* the mind is completely obscured and the respiration laboured and stertorous; except the functions of organic life all is in abeyance. In *syncope* consciousness is generally lost, and the condition of prostration is more transient.

The *causes* of collapse may be divided into those of sudden and those of gradual onset. When the condition is of sudden onset after injury or emotion it is usually described as shock.

(a) Of *sudden* onset:—(1) An overdose of chloroform or ether. (2) Surgical operations or severe injury. A vigorous man suffers more from operation than an old man or one who has been in bed for some time previously. Blows on the abdomen or extensive burns are always attended by more or less collapse. A relatively slight injury to a very sensitive part such as the testicle may produce collapse. (3) Severe and sudden emotion (terror or grief) or acute pain coming on suddenly, such as biliary or renal colic. (4) Poisoning by the narcotico-irritants (oxalic acid, carbolic acid, phosphorus, etc.) and by the asthenic poisons (hydrocyanic acid, aconite, digitalis, tobacco, veratria). A history in these cases may be absent. Ptomaine poisoning from tinned meats, etc., is attended by profuse diarrhœa. (5) Profuse hæmorrhage or diarrhœa, as in post-partum hæmorrhage and cholera. (6) Intestinal obstruction. (7) Perforation of some part of the alimentary canal with extravasation of its contents into the peritoneum. (8) Rupture of an abdominal cyst or of an abdominal or thoracic organ. (9) Pulmonary or other embolism. (10) Heat exhaustion after exposure to a very hot sun.

(b) Of *gradual* onset:—(1) Privation and exposure combined. (2) Profuse diarrhœa such as usually terminates lardaceous disease. (3) Peritonitis and other abdominal inflammation. (4) The asthenic types of fever, such as may attend enteric and yellow fever. (5) At the termination of many diseases described in the chapter on debility.

When a patient is found in a state of collapse or shock the physician has to diagnose the *cause* of the condition. After applying restoratives he should inquire, first, whether there is a history of injury or emotional disturbance, hæmorrhage, etc. Secondly, ask if the patient was in good health up to the time of onset of the condition of prostration, so as to exclude group b. Thirdly, inquire what food the patient has recently taken, and remember the possibility of poison. Fourthly, examine all the viscera, especially the heart and abdominal organs, beginning at the part which is or has been the seat of pain.

The immediate *treatment* consists in applying warmth to the body by means of hot bottles and warm blankets. The head should be lowered, the feet raised, especially in cases following external hæmorrhage. Stimulants, alcohol or ammonia may be given by the mouth if the patient can take them. Hypodermics of ether (♏ 20 to 60 every half-hour) or liq. strych. (♏ x.) or brandy should be given. Saline solution should be administered subcutaneously (§ 407) in cases where there has been profuse hæmorrhage or diarrhœa, and in many other cases of collapse.

GROUP II. MENTAL SYMPTOMS.

Disorders of consciousness and of the mind form a very large group. There are not many serious disorders of the nervous system in which mental symptoms do not appear sooner or later; and that complex organ the mind has also disorders peculiar to itself. These are generally omitted from text-books on medicine, but the scheme of this work would not be complete without a brief epitome of the more important.

It will be found convenient to deal with mental symptoms under four headings—

Sudden interruptions of consciousness—slight and transient, such as petit mal; severe and prolonged, such as coma §§ 524 and 525

Partial mental and "one faculty" defects, such as speech defects, or loss of memory . . . . § 527

Acute perversions of the mind, such as delirium and mania . § 530

Chronic perversions of the mind in adults, adolescents, and children, such as melancholia, dementia and various special types . . . . § 531 *et seq.*

[illegible]
MEDICO-CHIRURGICAL [illegible]

The patient complains of sudden transient **attacks of unconsciousness**—the case is probably one of SYNCOPE or EPILEPSY MINOR.

§ 524. **Sudden,** usually **brief and transient unconsciousness.** The patient comes perhaps complaining of "attacks," "sensations," "faints," "dizziness in the head," or "interruptions of thought." You have only the patient's account to guide you, and it may be a little difficult to diagnose the condition, but it is probably SYNCOPE, EPILEPSY MINOR, ARTERIAL (or senile) VERTIGO, AURAL VERTIGO, or MÉNIÈRE'S DISEASE.

It is necessary, first, to ascertain definitely whether the patient was really unconscious; did he know who was beside him, or hear when they spoke to him, and did he fall down? Secondly, the age and sex of the patient—epilepsy appears for the first time between 10 and 30, hysterical faints are almost always confined to young females. Thirdly, the history of previous attacks.

I. **Syncope** is a loss of consciousness more or less complete, generally of short duration, due to cardio-vascular failure of functional or organic origin. It does not generally last longer than a few minutes, though the duration and intensity vary considerably. Before the attack the patient is pale, sometimes a useful warning. The process of going off is accompanied by a disagreeable "swimming in the head," which has some resemblance to vertigo, or by an indescribable sinking feeling in the region of the stomach. The process of recovery is more gradual than petit mal. The diagnosis of cardiac syncope from nervous faints (of vaso-motor origin), and these from petit mal, and their causes **and treatment have been given in § 25, p. 47.**

The most interesting clinical features about the functional or "nervous faints" which occur chiefly in young women are as follows:—(1) Whereas the great majority of persons never faint, such attacks are liable to recur many times during the lifetime of certain individuals, with very little provocation. In other words, there seems an inherent or inherited weakness in that part of the vaso-motor apparatus concerned in some persons which does not exist in others. (2) The attacks are almost entirely confined to the female sex. (3) Such patients very frequently present the other "stigmata" of the hysterical diathesis (§ 519). (4) On inquiry, it will generally be found that these subjects are liable, on the occurrence of the same causes which produce the faints, to "flushes" or "flush storms," again indicating vaso-motor susceptibility, and that these flushes may be interchangeable with the faints. Thus a patient now under my care, æt. 27, is liable at all times to "flushings," but the same causes which determine these will, at the catamenial period, result in fainting. (5) There is invariably some determining cause of the attack, and this is generally of an emotional kind, such as fear, grief, fright or joy. (6) Copious urination of pale, highly watery urine very often follows the attacks, and seems, in my opinion, to indicate that dilatation of the arterioles of the splanchnic area has taken place. These and other considerations seem to point to the great probability that the essential cause of these attacks is weakness or susceptibility of the abdominal sympathetic.[1] We know that the blood pressure in the cerebral arteries is largely if not entirely regulated by the amount of blood in the splanchnic area, being high when the latter is relatively empty of blood, and low when it is relatively full.

II. **Epilepsy Minor** (syns. petit mal, epileptic vertigo). There are two varieties of idiopathic epilepsy (§ 592), E. major, consisting of unconsciousness with convulsions, and E. minor (petit mal), unconsciousness without convulsions. They sometimes alternate in the same individual. There are many degrees, shading from one to the other, and few dispute the identity of the two maladies. Only one circumstance can be seriously urged in this direction, namely, that amyl nitrite will often stop an attack of major, but increases the severity of an attack of minor, epilepsy. Epilepsy minor may be defined as a momentary or brief loss of consciousness; preceded—in about half the cases—by an aura or warning, unattended by convulsions, and often without falling, not followed by the stage of stupor, and the whole lasting rarely more than half a minute to a minute. In the attacks now under consideration, it may be that the patient only pauses in a conversation, or there is only a vacant look, dilated pupils, or momentary pallor of the face, which none but a close observer would notice. More usually, however, the patient—who generally refers to these attacks as "sensations"—feels giddy and loses his equilibrium for a few moments. The chief points of difference between nervous faints and minor epilepsy are given on p. 48. The absence of any apparent rotation of objects helps us to distinguish epilepsy minor from vertigo; and its appearance always for the first time in the earlier decades of life, distinguishes it from "senile syncope." Some say that petit mal is more likely to result in insanity than grand mal. *The treatment* is on the same lines as that of epilepsy major.

Masked Epilepsy is a still more incomplete form of epileptic attack, in which the patient, though unconscious, continues to perform automatically various acts during the seizure. Thus, a shoemaker under my care would continue his sewing; and Trousseau mentions a skilled violinist who continued to play with precision. He also mentions a judge who used to leave the Bench during an attack of masked epilepsy, micturate in a corner of his robing room and return to the Bench again without a return of consciousness. The criminal records show that homicide may be performed during these attacks.

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[1] Clinical Lecture on Hysterical Attacks, *The Lancet*, July 20, 1901.

III. **Senile Syncope; Senile or Arterial Vertigo; Apoplectiform Attacks.** Under these terms may be included various attacks attended by unconsciousness, varying in degree from a transient interruption of thought to a severe loss of consciousness. Those who have much to do with old people are aware that brief losses of consciousness are extremely frequent among them. Sometimes it amounts to nothing more than a momentary confusion of thought. They were variously described by my old patients in the Paddington Workhouse as "swimmings," or "dizziness," "giddy faints." They often gave rise to a momentary reeling if they happened to be walking at the time. But sometimes it is a definite unconsciousness, lasting one or two minutes, or longer, in which they fall unless they lean against or catch hold of something. Every gradation is met with between these attacks and a severe syncopal or apoplectic seizure. These attacks are generally associated with arterial hypermyotrophy[1] or some other form of arterial disease, especially if this be combined with cardiac failure. In a few instances of the severer form of attack, there were definite evidences after death of minute softenings or hæmorrhages of various dates, sometimes in considerable numbers, with which the attacks were undoubtedly connected.

IV. **Aural Vertigo** (§§ 510 and 613). Patients often speak of attacks of aural vertigo as "faints," though they are not usually attended by unconsciousness. It is not, however, sufficiently recognised that severe attacks of aural vertigo may be attended by complete loss of consciousness of some duration, and that such attacks may be connected with middle ear disease as well as lesions of the inner ear.

One of the most severe cases of paroxysmal vertigo with unconsciousnses, due to middle ear catarrh, which I have met with, occurred in G. C. B., æt. 38, a cattle dealer, who consulted me in February, 1896, for severe attacks of giddiness, which he had had for 2½ years. The giddiness would come on suddenly, without warning, "like a windmill in the head," and "things went all round," sometimes he became unconscious and fell down unless near something to catch hold of, sometimes they were followed by nausea or vomiting. He had lately had two or three a week, and since August, 1894, he had had to give up work. In one very bad attack he became quite unconscious for some time and fell out of his cart. Six months before the vertigo had come on, January, 1893, his hearing had gradually become defective, and ever since then he had had a sensation as of a "kettle singing in his head." On examination, I found his hearing to be about half normal, but the perosseous hearing was good. All his symptoms were attributable to middle ear catarrh. After six months' pharyngeal and aural treatment under Dr. Bolton Tomson (of Luton, Beds), his hearing was quite restored, and he was able to leave off the bromides without a return of any of his troublesome and dangerous attacks.

§ **524a. V. Ménière's Disease** (or labyrinthine vertigo), may be defined as a paroxysmal vertigo, accompanied by a transient loss of consciousness, associated with nerve deafness due to hæmorrhage into the vestibule or semi-circular canals, supervening suddenly with an apoplectiform attack. The term is sometimes applied to any form of vertigo associated with deafness, but this does not tally with Ménière's original description,[2] and the name is better exclusively reserved for vertiginous attacks attended by loss of consciousness, and associated with deafness.

There are four classical symptoms. (i.) The vertigo is always in paroxysms in which, after the first attack, consciousness is retained though the patient may fall, owing to the disturbance of equilibrium. Irritation of the left semi-circular canals usually produces a sense of rotation from left to right; destruction the converse. (ii.) The attacks are commonly attended by nausea, or even vomiting. (iii.) There is deafness in the great majority of cases, on one or both sides, the patient being unable to hear a tuning fork placed on the teeth or the head (perosseous, *i.e.*, nerve deafness); and (iv.) tinnitus, or buzzing in the ears. In characteristic cases of Menière's disease, these four symptoms supervene suddenly with an apoplectiform attack (accompanied by transient loss of consciousness) which has been shown to be due to hæmorrhage into the labyrinth. (i.) and (ii.) afterwards become paroxysmal, (iii.) and (iv.) more or less permanent. Knapp[1] has observed that the hearing is impaired at first only for the higher and lower octaves; and Charcot states that some patients have the vertigo and buzzing only while the deafness is partial, disappearing when this becomes total; this is probably diagnostic of labyrinthine as distinguished from central vertigo.

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[1] Trans. Path. Soc. Lond., 1904.

[2] It seems that all Méniére's original cases came on in an apoplectiform manner. *Gazette Médicale*, 1861.

In regard to *treatment*, the disease is undoubtedly very intractable, if not incurable, and our efforts should be directed mainly to the circulatory system, so as to regulate it and avoid a repetition of hæmorrhage. The symptoms may be relieved by bromides, and other sedatives. Charcot recommends grs. v. of quinine thrice daily, and he mentions one case which was cured by this means. Trinitrin often relieves the symptoms for a time.

The patient has sudden, more or less prolonged, and **complete unconsciousness**—the case is one of apoplectic or other form of COMA.

§ **525.** **Coma** is a condition of loss of consciousness coming on more or less suddenly, in which, in its complete form, all signs of vitality excepting those of organic life are suppressed. The patient is deprived of all power of movement and sensation. He is neither able to hear nor to understand an order to put out his tongue shouted into his ear. The limbs fall helplessly into any position. The eyelids are closed, and the conjunctival reflexes absent. The respiration is slow and stertorous, owing to the flapping of the palate and the falling back of the tongue on to the posterior wall of the pharynx. The pulse and the respiration are the only signs of life. The temperature is, at any rate at first, normal or sub-normal. The *typhoid state* (§ 347) is a term applied to the comatose condition which supervenes in certain fevers, and may be recognised by the presence of pyrexia, the history, and muttering delirium. SYNCOPE is readily distinguished from coma by the unconsciousness being much less in degree and much more transient. The following is a list of the possible causes of coma:—

I. **Head injury.**
II. **Apoplexy and other vascular lesions.**
III. **Other gross and functional cerebral lesions.**
IV. **Alcohol.**
V. **Opium.**
VI. **Uræmia.**
VII. **Diabetes.**
VIII. **Cholæmia.**
IX. **Heatstroke.**
X. Addison's disease. Raynaud's disease, and other rare conditions.

Coma in children arises under somewhat different conditions (§ 526).

The CLINICAL INVESTIGATION of coma is of the highest importance, as it is an emergency of the gravest significance. When called to such a patient, whom one sees perhaps for the first time, the question as to the cause is one of the most difficult that we have to solve. The commonest causes of such a condition are drink, cerebral apoplexy, head injury, opium poisoning, and uræmia, and the prognosis and treatment differ in these several conditions. Your mode of procedure should be as follows :- (1) *Examine the head* (and other parts) carefully to see if there be any signs of injury, and if the case be a medico-legal one, make a precise written note on this point. If there be evidence of an injury, the question of whether the injury has caused the coma, or whether it occurred after the patient was seized, should be borne in mind. (2) Note the *odour of the breath*. If this be alcoholic, it does not follow that the condition is due to drink, for stimulants are frequently given by the friends to restore the patient during such an attack. Note also whether the breath has the sweet odour of diabetes. (3) Observe most particularly the state of each *pupil*, and the conjunctival reflex. Both pupils are much contracted in opium poisoning and hæmorrhage into the pons. Hæmorrhage into other and commoner situations within the cranium usually causes inequality or dilatation of both pupils. In the other causes of coma they are usually both dilated. (4) Ascertain whether any *paralysis* of the limbs is present, or whether the face is drawn to one side. This may be a little difficult, but usually in apoplexy the leg and arm of one side of the body, opposite to that of the lesion, are very rigid, or very flaccid, as compared with the other side, and by raising the limbs and allowing them to fall, it will easily be found whether one side is more rigid or more flaccid than the other. In nearly all cases due to gross cerebral lesion (tumour, abscess, meningitis, etc.), one side is weaker than the other. (5) Count the *pulse*, examine the *arteries*, and auscultate the *heart*. High arterial tension suggests uræmia or apoplexy ; great slowness suggests opium poisoning ; a presystolic murmur suggests cerebral embolism. (6) Count and observe the *respirations*. A stertorous or snoring quality is simply an indication of the profundity of the coma, being due to paralysis of the tongue and palate. The respirations are very slow in opium poisoning. In grave cases of apoplexy and uræmia, the respiration assumes a Cheyne-Stokes character. (7) Take the *temperature*. It is often very low in uræmia and in opium poisoning, sometimes a little lowered in apoplexy and in drink. There is sometimes a tendency for it to run up after an attack of apoplexy, and this is of very serious import. (8) Procure (by catheter if necessary) and examine some of the *urine*. The absence of albumin is against renal disease ; a small amount of albumin does not help one much to distinguish between apoplexy and uræmia. In diabetic urine we find sugar ; in opium poisoning, morphia. Atropine and other vegetable alkaloids are also excreted by the urine. (9) Observe the *age*, and inquire into the *history*—whether the attack came on suddenly in apparent health, or after some previous indisposition. As regards the age, coma in childhood is almost confined to post-epileptic coma, meningitis, cerebral tumour or sinus thrombosis ; about middle age, cerebral hæmorrhage is to be suspected. Coma sometimes arises in the course of a disease the history of which is readily revealed by inquiry ; such, for instance, as epilepsy. But, on the other hand, the patient's friends may have been quite unaware of the existence of any disease, such, for instance, as diabetes, contracted granular kidney, or cerebral tumour in the frontal region.

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[1] "Archives of Ophthalmology and Otology," 1871, Knapp and Brunner.

The commonest causes of coma in every-day practice are drink, intracranial hæmorrhage (with or without injury), opium poisoning, and uræmia—see table below.

TABLE OF DIAGNOSIS OF COMA DUE TO DRINK, APOPLEXY, OPIUM POISONING, AND URÆMIA.

	Pupils (P.) and Conjunctival Reflex (C. R.)	Pulse and Respiration (R.).	Paralysis.	Depth of Coma.	Course.
Drink.	P. equal, normal, or dilated. C. R. present.	*Pulse rapid and strong, then weak.* R. Normal or snoring.	None, but inco-ordination if able to walk or move.	*Can be roused.*	Progressive recovery in 12 hours.
Apoplexy or Fracture with Compression.	P. usually dilated and unequal. *C. R. lost.*	Pulse generally full and bounding. R. stertorous; may be Cheyne-Stokes.	*Hemiplegic* rigidity or flaccidity and often *facial paralysis.*	Cannot be roused.	Stationary.
Opium Poisoning.	*P. very contracted and equal.* C. R. usually present.	Pulse and R. both very slow. R. stertorous.	General weakness.	Can sometimes be roused.	Progressive towards death or recovery in 10 to 12 hours.
Uræmia.	P. normal or dilated. C. R. usually present.	*Pulse slow and arterial tension high.* R. sighing, and may be Cheyne-Stokes.	None.	Cannot be roused.	*Coma alternates with convulsions.*

1. **Head Injuries** may produce either *concussion* (bruising) or *compression* of the brain; the symptoms of which differ more in degree than in kind (table below). Injury may also be accompanied by conjunctival hæmorrhage, paralysis of the cranial nerves, and inequality of the pupils. Bleeding from the ear and sub-conjunctival ecchymosis do not necessarily (though usually) indicate fracture of the base. Injury to the head may produce compression of the brain, and therefore coma, in four ways: (1) fracture of the skull, with depression; (2) hæmorrhage into or upon the brain; in both of which the coma comes on immediately after the injury. (3) The effusion of inflammatory products, when the coma comes on after an interval of a few days; and (4) abscess, when coma comes on after a week or two at least.

TABLE OF SYMPTOMS OF CONCUSSION AND COMPRESSION.

Concussion of the Brain.	*Compression of the Brain.*
Symptoms—As in shock (*q.v.*), *plus* sudden unconsciousness, but patient can be roused in most cases.	Completely unconscious, cannot be roused.
Pupils—Equal, dilated, sluggish reaction to light.	Immobile, often unequal, at first contracted, later on dilated.
Respiration — Shallow, slow, sometimes sighing.	Slow, stertorous, sometimes irregular.
Motor System—Muscles relaxed, but no absolute paralysis.	Paralysis; cheeks blown out with expiration; often rigidity on one side of the body.
Bladder—Frequent micturition.	Retention till overflow with "false" incontinence.

NOTE.—There is considerable difficulty of diagnosis when a case has *slight compression* or a *very grave degree of concussion.*

§ 525a. II. **Apoplexy** (a "stroke") is a term which may be conveniently retained to indicate a sudden unconsciousness due to a vascular lesion within the skull. There are three kinds of such vascular lesions—hæmorrhage, embolism, and thrombosis. The older authors used to state that hæmorrhage from the rupture of a cerebral artery could be distinguished from simple embolism or thrombosis by the occurrence of *loss of consciousness* (sometimes accompanied by convulsions) in the former, and not in the latter. But further experience has shown that this distinction is only a matter of degree—profound coma arising from the embolic blocking of a moderately large artery, and also from thrombosis; while, on the other hand, slight hæmorrhage supervening gradually may be unattended by loss of consciousness. The *extent* and the *suddenness* of the vascular lesion, rather than its nature, determines the presence of coma.

Symptoms of cerebral hæmorrhage. A prodromal stage or warning in the form of headache or vertigo for some days before may be complained of, and is undoubtedly connected with the high arterial tension which is its leading etiological factor. The coma may come on very suddenly without warning, followed or not by convulsions. Sometimes the paralysis comes on with faintness and vertigo only; or the paralysis may come on more gradually, followed later by unconsciousness (ingravescent apoplexy). Sometimes it comes on during sleep. The lesion causes paralysis on one side of the body, which is indicated either by a greater rigidity or a greater flaccidity, or by the absence of spontaneous movements on that side. The pupils are unequal, the conjunctival reflex lost, and the temperature, particularly in large hæmorrhages, is usually at first one or two degrees below normal. In the course of 24 to 48 hours the thermometer usually shows a rise of one or two degrees, at which point it remains for several days. A rapid elevation of temperature within a few hours of the seizure indicates hæmorrhage at the base, and therefore a speedily fatal termination.

Diagnosis. In view of the importance of differentiating this condition in an emergency, a table is given above (p. 938). It should be remembered that apoplexy frequently supervenes in the course of chronic Bright's disease, and therefore uræmia and apoplexy may be concurrent. The diagnostic features of the greatest value in apoplexy are the state of the pupils, particularly their inequality, the loss of the conjunctival reflex, and the presence of hemiplegia. The diagnosis of the various *forms of vascular lesion* is given in a table below.

TABLE OF THE DIAGNOSIS OF CEREBRAL HÆMORRHAGE, EMBOLISM, AND THROMBOSIS.

	Cerebral hæmorrhage.	*Embolism.*	*Thrombosis.*
Age.	Middle and advanced age.	Any age, but frequently young.	Any age.
Causes.	1. Arterial degeneration. 2. Vascular strain. 3. Excitement.	1. Cardio-valvular lesions, especially mitral stenosis. 2. Thrombus in the peripheral vessels.	1. Syphilitic endarteritis. 2. Cerebral atheroma. 3. Exhausting disease; phthisis; anæmia. 4. Slowing of blood *e.g.*, cardiac enfeeblement.
Onset.	Coma usually sudden, sometimes with convulsions.	Sudden onset of paralysis, but usually no loss of consciousness or convulsions.	Paralysis may be sudden, or after premonitory symptoms, vertigo, convulsions. Coma unusual.

As regards the *locality* of the hæmorrhage the usual position (about 76 per cent.) is the *internal capsule*, giving rise to hemiplegia of face and body on the side opposite to the lesion. In about three-fourths of the cases of hæmorrhage into the *ventricles*, there is paralysis or rigidity of all four limbs, and the condition is uniformly fatal. Marked contraction of both pupils, or crossed hemiplegia, suggests hæmorrhage into the *pons*: hurried or Cheyne-Stokes respiration is more common with hæmorrhage in this position and the prognosis is grave. *Meningeal hæmorrhage* is suggested by the absence of definite paralysis or the presence of initial and recurring convulsions. *Pachymeningitis hæmorrhagica* (hæmorrhage into the thickened meninges) is a condition giving rise to attacks of coma, which differ (in most of the cases I have seen) from the other causes now under consideration in their slow advent. It far more often occurs in lunatics, and in the dementia of old age. *Conjugate deviation of the head and eyes* towards the paralysed side is frequent when the hæmorrhage involves the motor tract.

Prognosis. About half the cases of apoplexy, taking all cases together, recover from the attack, but with remaining paralysis. The depth and duration of the coma is a fairly certain measure of the extent of the mischief, and therefore of the prognosis. The signs indicating deep coma, which are therefore of unfavourable import, are loud stertor, completely insensitive conjunctivæ, flapping cheeks, and increasing cyanosis. Convulsions, or the early appearance of rigidity or a sudden rise of the temperature are unfavourable. Coma coming on slowly, and progressively increasing (that is, ingravescent apoplexy) is more unfavourable than that which comes on more suddenly, with less complete coma. A more unfavourable form still is when, shortly after the first attack of apoplexy, a second supervenes; from this the patient rarely recovers. As regards *locality*, the worst positions are intra-ventricular hæmorrhage, basal hæmorrhage, and hæmorrhage into the pons.

Etiology. Cerebral hæmorrhage is more frequent in the male sex, and in those over 40 or 50. The rarer cases of "apoplectic seizure" in a person under 40 are almost invariably due to embolism or thrombosis. Heredity plays an important part by reason of the tendency to vascular disease which runs in families. There is a marked predisposition in plethoric persons to suffer from cerebral hæmorrhage—the stout, thick-necked build, with ruddy cheeks. Disease of the vessels is an almost necessary precursor to their rupture. High arterial tension is a most important factor in the causation of apoplexy; it predisposes to arterial disease, and may also determine the hæmorrhage. The causes of high arterial tension are given in § 64; but the commonest cause of high tension is chronic Bright's disease, and that is why this morbid condition is so frequently associated with cerebral hæmorrhage. Leukæmia, purpura, and other blood diseases may occasionally cause cerebral or meningeal hæmorrhage.

Cerebral embolism. The preceding remarks have reference to cerebral hæmorrhage, but cerebral embolism involving a fairly large artery may give rise to all the symptoms of apoplexy. An "apoplectic stroke" in persons under 40 is almost invariably due to embolism or thrombosis, though hæmorrhage may occur in children. The age of the patient, the presence of cardiac disease or some other condition giving rise to embolism, aids us in diagnosis (see table, p. 940).

Thrombosis of the Cerebral Arteries is also a cause of apoplectic seizure. In most cases thrombosis arises from a *gradual* occlusion of the lumen of a vessel by chronic arterial disease in the aged, or by syphilitic endarteritis in the young. The supervention of symptoms, however, is usually sudden, and in only a proportion of cases attended by coma (see table above).

Thrombosis of the Cerebral Sinuses is not common, but may give rise to coma and all the symptoms of apoplexy. It may arise from caries of the skull of syphilitic or tuberculous origin, extension from a cerebral abscess, and occasionally from the pressure of an aneurysm, gumma, or other tumour; or in association with meningitis. *Non-pyogenic thrombosis* (especially of the superior longitudinal sinus) also occurs with cachectic conditions, chronic diarrhœa, enteric fever, and marasmus in children. *Septic thrombosis* and the differential signs of thrombosis of the lateral, cavernous and longitudinal sinuses are described under intracranial inflammation (§ 544).

The *prognosis* of cerebral embolism as regards life is usually good, but if the causal condition remains it is apt to recur. In thrombosis, the prognosis is good when due to syphilitic endarteritis, less favourable when occurring in the aged, and extremely grave when associated with exhausting disease and anæmia.

The *treatment of an apoplectic seizure* is not very hopeful. Perfect rest and quiet are very important. The patient should, as a rule, be left in the room where the seizure occurred—a mattress being placed on the floor, if necessary, rather than incurring the movement necessary to raise him on to a bed. The head and shoulders should be raised, and the patient turned gently over to one side to prevent the tongue falling back into the pharynx.

The administration of food is, as a rule, undesirable, at least by the mouth, for fear of it passing into the air passages; and alcohol must be absolutely forbidden. The bladder should be watched, and the catheter carefully passed if necessary. The patient will do no harm for a day or two without nourishment by mouth, and the lips may be moistened by a feather dipped in water. In cases due to *embolism* nothing further can be done excepting to prevent a recurrence; in *thrombosis* (other than syphilitic) stimulants are indicated. In *hæmorrhage* a brisk purge should be given; two drops of croton oil or 4 to 8 grs. of calomel on the tongue is a good method, followed if necessary by an enema of castor oil or turpentine. The chief indication is to prevent any extension of the hæmorrhage. If the pulse is full and bounding, it is a good practice to bleed to the extent of 3, 5 or even 10 ounces; and these patients nearly always do well. The administration of aconite or veratrium viride has been suggested to reduce the blood pressure in lieu of venesection. An ice-bag, or a cooling lotion to the head is recommended by some, and may be of some benefit if care be taken that after it is once started there is no intermission, for the reaction in such an interval may increase the mischief by determining blood to the head. Blisters to the back of the neck, and mustard plasters to the calves or soles of the feet "to rouse the patient" are in my belief worse than useless. Hæmostatics, ergot, gallic acid, acetate of lead have been recommended, and belladonna may be of use.

III. **Other gross and functional cerebral lesions.** Generalised convulsions are always attended by some disturbance of consciousness, and sometimes by coma, during and sometimes following the attack. But in such circumstances the convulsions constitute the major feature, and their causes are therefore given under that symptom (§ 591). It will be seen that the list of these causes corresponds very closely with the causes of coma, and this is what one would expect; for in general terms cortical *compression* is manifested by coma, cortical *irritation* by convulsions, and toxæmia may be evidenced by either. If the history is wanting difficulty in diagnosis may be experienced. Among the chief causes of both convulsions and coma are the following.

1. In POST-EPILEPTIC STUPOR the unconsciousness is not so complete as in apoplexy, there is no hemiplegia, and within a few hours the

patient wakes. There is a history of previous attacks if the patient is an adult.

2. CEREBRAL TUMOUR and ABSCESS may give rise to attacks of coma which, in the absence of a history of previous ill-health, are difficult to distinguish from apoplexy. In such gross lesions of the brain there is (1) optic neuritis; (2) paralysis of the cranial nerves and perhaps hemiplegia. Tumour of the frontal lobe, however, may cause no paralyses. (3) The coma not infrequently alternates with or is attended by convulsions. (4) A history of headache, giddiness and vomiting may be present.

3. In one-fifth of the cases of DISSEMINATED SCLEROSIS attacks of coma occur (Charcot), lasting a day or two, and then passing into a state of stupor; in these the face is flushed, pulse rapid, and temperature elevated to 104° or 105°. The tremor and other symptoms are always worse as the patient emerges from these attacks.

4. GENERAL PARALYSIS OF THE INSANE at some stage is almost invariably accompanied by fits of various kinds; sometimes they are comatose, sometimes epileptiform with partial or complete loss of consciousness, and sometimes without unconsciousness. Unconsciousness is a very bad sign, and the patient frequently dies in such attacks.

5. Certain ACUTE and SUBACUTE CEREBRAL LESIONS such as tuberculous and simple meningitis, cerebro-spinal meningitis and septic sinus thrombosis may cause coma. Coma in such conditions is usually of late onset, due to compression of the brain, and a history and other signs are obtainable (see intracranial inflammation, § 510). However, in tuberculous meningitis coma may be of sudden onset, especially in children.

IV. **Intoxication** or acute alcoholic poisoning. The coma which supervenes after heavy drinking, or a single large dose of alcohol, may very closely resemble apoplexy and cerebral compression from head injury. In any case of doubt it is wise to admit the patient to the hospital, or to treat him on the supposition of the more serious condition. If house surgeons would bear this in mind, we should see less of those paragraphs in the newspapers headed "drunk or dying." The smell of alcohol in the breath is fallacious, as friends may have given alcohol to restore the patient. The chief differential features of the coma of intoxication are (1) the coma is rarely so profound as in apoplexy, and the patient can generally be roused. (2) The absence of inequality of the pupils, hemiplegia, or convulsions (see table, p. 938). (3) Procure some of the urine, and add one or two drops of the urine to 15 drops of a chromic acid solution, made by adding one part by weight of potassium bichromate to 300 parts by weight of strong sulphuric acid. The solution turns a bright emerald green if alcohol be present in quantity (Anstie).

V. In **opium poisoning** the patient becomes progressively drowsy, and coma succeeds gradually; (2) the pupils are equal and extremely contracted; (3) the pulse and respirations are slow; (4) there is no hemiplegic rigidity or flaccidity of the limbs; (5) the coma gradually deepens, the face becomes cyanotic, and the pulse and respiration gradually cease together.

For the symptoms of poisoning by other narcotic drugs, the reader is referred to manuals on toxicology, but it is well to remember that chloral and chloroform, coal-gas, belladonna, the bromides, cannabis indica, and occasionally hydrocyanic acid, may all act as narcotic poisons.

VI. **Uræmia** ("serous apoplexy" of older authors[1]) may lead to coma in the advanced stages of kidney disease. (1) The coma is rarely quite so profound as in apoplexy; it is more of a stupor or drowsiness, which gradually deepens. (2) In the great majority of cases, stupor alternates with convulsions; and in many cases it alternates with muttering delirium. (3) There is an absence of hemiplegic rigidity or paralysis. (4) There is albuminuria. (5) If a history is obtainable, the earlier symptoms of uræmia will be revealed (§ 273).

VII. **Diabetic coma** supervenes very suddenly, often in apparent health, and is very profound. Its two characteristic features are (1) the sweet odour of the breath, somewhat resembling that of chloroform, and (2) the presence of sugar in the urine. It almost invariably results in death, being one of the most frequent modes of termination in that disease.

VIII. **Cholæmia.** Hepatic diseases which result in destruction of the secreting tissue of the liver—notably the later stages of cirrhosis and acute yellow atrophy—give rise to a condition clinically resembling uræmia. Pathologically also the resemblance holds, for one of the functions of the liver is to elaborate those nitrogenous products which it is the function of the kidney to excrete. Clinically this condition is, in most cases, differentiated by the jaundice, slight in the concluding stage of cirrhosis, very marked in acute yellow atrophy. If jaundice be absent, it can only be identified by the hepatic symptoms (*e.g.*, ascites, etc.), and the circumstances under which it occurs. The coma under these circumstances has been called cholæmia, under the impression that it is due to the presence of bile in the blood; but bile freed from mucus injected into the blood does not give rise to symptoms.

IX. **Heat-stroke** (Sun-stroke) is classified into the "cardiac" variety, in which the patient suddenly goes off into a dead faint, with symptoms pointing to failure of respiration and circulation; and the "cerebro-spinal" variety, in which coma gradually supervenes. The circumstances under which it occurs are the only means of its differentiation. The comatose form is described in § 376.

X. **Certain rare diseases**, *e.g.*, **Addison's disease** and **Raynaud's disease**, are occasionally attended by sudden coma, perhaps with vomiting and hemiplegia. A malignant form of **malaria** (§ 379), is attended with coma which, coming on suddenly, may lack a history; and English trained medical men newly arrived in India may mistake these cases for apoplexy. *Excessive muscular exertion* has caused coma, probably due to accumulation of toxic products (Dr. V. Poore, *The Lancet*, 1894, vol. i., p. 1066).

The *prognosis* of coma is always grave, and the gravity increases with the depth and the duration of the coma. The coma after head injury usually comes under the care of the surgeon. The coma of apoplexy and other vascular lesions has been already dealt with. In post-epileptic coma if the patient does not recover within a few hours the status epilepticus is present; such a condition being very rare, the diagnosis should be carefully reconsidered. Coma due to degenerative conditions of the nervous system is usually recovered from, leaving behind perhaps a temporary paralysis. Coma occurring with tumour of the brain or acute lesions is usually fatal. The prognosis of opium poisoning depends upon the time which elapsed before the patient was seen, the treatment adopted and the vigour with which it was carried out. Uræmic coma is not so unfavourable as might be thought; cases recover with proper treatment, but sooner or later the condition recurs. In diabetic coma the patient rarely rallies.

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[1] The older authors recognised the clinical resemblance of uræmic coma to the coma of cerebral apoplexy, but they found no intracranial hæmorrhage. In cases of chronic Bright's disease the cerebral convolutions waste, and the space is occupied by the effusion of serum on the surface of the brain, which was therefore regarded by them as the cause of coma, hence the term "serous apoplexy."

Treatment. Apoplexy has been already dealt with, and the treatment of head injury is carried out on similar lines, bearing in mind that surgical aid is necessary in many cases. For alcoholism and poisoning a prompt emetic should be given, such as zinc sulphate, grs. 30, mustard and hot water or salt and water; a hypodermic injection of apomorphine acts more promptly and is one of the best remedies for intoxication. Opium poisoning is treated by frequent washing with the stomach-pump; coffee, atropine or strychnine are given if the heart or respiration be failing. The patient must be kept awake, never allowed to doze, by walking him about and applying electricity to the limbs; ammonia to the nostrils, warmth to the extremities and artificial respiration will generally pull him round. For uræmia eliminate the poison in the blood by brisk purgatives, hot packs, venesection and saline injections.

§ **526. Coma in children**, apart from injury, may be due, in order of frequency, to tuberculous meningitis, post-basal meningitis, suppurative meningitis, cerebral tumour, syphilitic pachymeningitis, sinus thrombosis, and hæmorrhage; diabetes, abscess and cysts are rare causes. The history, mode of onset, and associated symptoms aid the diagnosis. Tuberculous meningitis is by far the most frequent cause; out of 86 cases of coma in childhood Dr. F. E. Batten found tuberculous meningitis in 50 cases, non-tuberculous meningitis in 17, and cerebral tumour in 16 cases. Cerebral hæmorrhage occurs chiefly in association with the specific fevers, such as small-pox and whooping-cough, also with rickets and scurvy. In marasmic conditions thrombosis of the longitudinal sinus (§ 544) may ensue, together with meningeal hæmorrhage, giving rise to convulsions followed by coma. Thrombosis of the veins of Galen (§ 621) and lateral sinus thrombosis which occurs with ear disease (§ 544) may cause coma.

GROUP II. β. *Partial mental and "one faculty" defects.*

One faculty of the mind such as the memory or Attention may be affected, and there may also be various partial mental defects which do not amount to insanity, though they may constitute its earlier phases. These will now be considered—

Defects of speech (motor and mental).
Defects of memory (partial and entire).
Defects of attention (deficiency and excess).
Defects of other mental faculties.
Hypnotism.
Catalepsy and trance.

§ 527. **Defects of speech and other signs of thought** may be purely motor, or they may be of central or mental origin (ideo-motor or ideo-sensory), but it will be convenient to consider them both here as they are so frequently associated. All of these defects may be simply and graphically represented in the following scheme :—

Defective communication with others.

Defects in the **outgoing** processes .	motor defects (paralysis)	
	ideo-motor . . .	speech. writing.
Defects in the **incoming** processes .	ideo-sensory . .	visual. auditory.
	blindness or deafness	

Motor defects of speech are recognised by an **error in articulation** : the patient says the words but pronounces them badly, just as in cases of motor defect of writing the patient can sometimes write but writes badly. The muscles of speech are those of the lips, tongue, palate, larynx and respiration, and these, like other muscles, may be the seat of paralysis, tremor, spasm, or inco-ordination.

Clinical Investigation. In order to investigate a case of motor defect of speech, we must first pay attention to each of the different muscles just named. In the course of conversation with the patient we can generally detect any of the motor defects which are mentioned below. If not, we may ask the patient to repeat the alphabet through, and we shall readily detect any paresis or other defect of the muscles. (2) Notice whether there be any tremor of the lips or of the tongue when protruded ; any syllabic utterance; or, on the other hand, any slurring or running together of the words. (3) The presence of any hemiplegia, facial paralysis, or other paralysis should be noted. (4) If all these tests be negative, and still the patient is unable to communicate his thoughts properly, turn to mental aphasia, § 528.

1. **Paralysis of the muscles** of the lips, face, and tongue is met with in most cases of *hemiplegia* in the early stage. Here one side of the face is generally obviously affected, especially its lower part, and the tongue when protruded deviates to the paralysed side. The speech is thick, and often quite unintelligible. But as the face and tongue recover, the speech returns. This is a pure defect of the muscles of articulation.

2. **Partial paralysis** of the muscles of articulation is met with in its most typical form in the case of *bulbar paralysis* (glosso-labio-laryngeal palsy) and pseudo-bulbar paralysis. At first, when the tongue is chiefly affected, difficulty occurs with such letters as L, R, N, T, and there is an

obvious difficulty in pronouncing words, so as to give the impression of the tongue being too large for the mouth. Soon after this, the labial letters also give rise to difficulty (as P, B, M), and the palate becomes involved, the speech becoming nasal, difficulty occurring with guttural letters—K and G (hard). Towards the end of this affection, speech is totally lost (alalia), and the patient can only utter meaningless grunts.

3. **A fine tremor of the lips** with **slurring speech** is very characteristic of *general paralysis of the insane.* The syllables are run together as in intoxication, "British Constitution" becomes "Brish Conshishune." It is also met with in delirium tremens, some cases of meningitis, and occasionally in disseminated sclerosis.

4. **Syllabic or staccato speech** is more characteristic of *disseminated sclerosis* than the preceding; there is a pause between each syllable, and each syllable of a word is equally accentuated (con-sti-tu-shon), as in scanning Latin verse, hence sometimes called "scanning speech." Occasionally this kind of speech is met with in tabes dorsalis.

5. **Stammering** is a spasmodic disorder of the muscles of articulation, and sometimes of respiration. It is somewhat like the defect of chorea, but if carefully noticed, it will be seen that the spasm is more of a tonic than of a clonic character, and it lasts indefinitely. Most difficulty is experienced with words commencing with explosive or labial letters—B, D, P, T, K, or G. Excepting in bad cases, the patient can whisper or sing without a defect. In severe cases the spasm tends to spread to other muscles of the face, or other parts; the patient, for example, remaining with his mouth wide open, or his face screwed up into some contortion for an appreciable time before any sound is uttered. The affection appears in childhood, and there is very generally some hereditary or other manifestation of defect in the nervous system. *Syllable stumbling* is a variety of stammering in which one or more syllables of a word are repeated. The curability of stammering depends a good deal upon its previous duration, and the age of onset. It is worse when it comes on in the adult. The *treatment* is a matter for very careful education, and there are some very good teachers who make the cure of it their speciality. Much can be done by teaching the patient to manage his breath, and always to take a deep breath before starting to speak. Or, again, I have frequently been able to assist such patients by instructing them to beat time with their hands, and, without actually singing, to adopt a sing-song method of speech.

6. **Lalling** or infantile speech is that in which the letters difficult to pronounce—*e.g.*, R, L, C, Sh—are avoided; British is pronounced Bitty.

7. In **idioglossia** the child has a speech of his own, which is unintelligible excepting to those accustomed to the child. It is due to a defective power to reproduce *the sounds* of words said to him. He has to be taught by a system of lip reading.

8. **Rhinolalia operta** is the speech met with in cleft palate and paralysis of the soft palate (*e.g.*, diphtheritic) in children. In it the speech has an unmistakable nasal quality. In **rhinolalia clausa**, on the other hand, which is due to a spasm or stiffness of the soft palate, the normal nasal quality of the speech is wanting, and this produces the somewhat affected style met with in some hysterical cases.

§ **528. Aphasia** is, as Trousseau aptly put it, loss of memory for the signs of thought. In some cases the patient's speech and power of writing are so much deranged that he cannot communicate his ideas to you; in other cases you are unable to communicate with the patient because he cannot recognise words spoken to or written for him.

For speech and writing, the two **outgoing** processes by which we communicate to others, two structures must be intact and their functions normal—

(1) The motor *apparatus* of speech and writing, and

(2) Initiating *centres* in the cerebral cortex which have been gradually educated up to the art of speaking and writing. These centres are appropriately called **ideo-motor** centres, and contain a series of educated recollections of the complicated movements necessary to produce speech or writing.

For the recognition of spoken or written language, the **incoming** processes of communication, two parts also are necessary (Fig. 157)—

(1) The sensory *tracts* of vision and hearing, and

(2) Certain receptive *centres* in the cerebral cortex, **ideo-sensory** centres, educated up to the art of recognising and naming words heard and things and words seen (see tables, p. 950 and p. 951).

In mental aphasia we are concerned solely with the ideo-motor (or outgoing) and the ideo-sensory (or incoming) centres in the cerebral cortex. Defects of the motor apparatus of speech have been fully considered (§ 527), and defects of the organs of vision and hearing are given elsewhere; in what follows we must assume that both of these are healthy.

(a) **Ideo-motor aphasia.** Are the patient's powers of **speech** or **writing** defective? We will first consider the out-going forms of aphasia: aphemia (ideo-motor defect of speech) and agraphia (ideo-motor defect of writing).

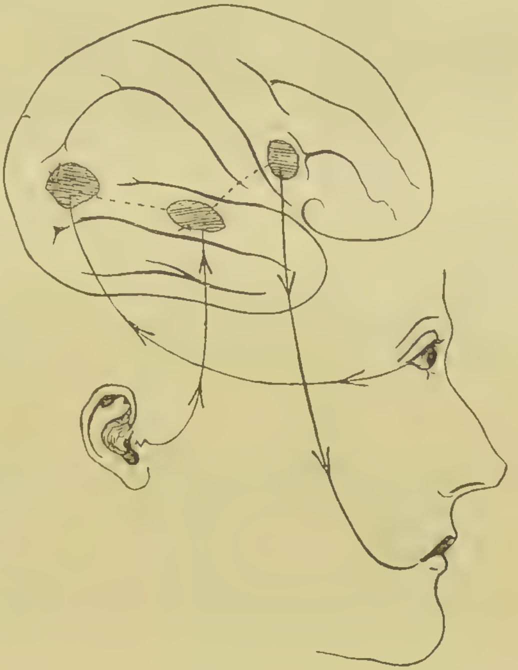

Fig. 157.—Apparatus of Speech (represented in error in the right cerebral hemisphere). The auditory and visual ideo-sensory centres and the ideo-motor centre of speech.

The commonest instances of this form of aphasia are associated with right hemiplegia. In certain cases of hemiplegia we find after a time that although the patient recovers the use of his lips, tongue, and arm, he is still unable to name things properly or to converse, except by writing or by pantomime. This disability may exist in many degrees; in some cases the patient only occasionally says a wrong word, in others he cannot use a single word correctly. In some of the worst cases the patient makes use of one or two words only (recurring utterances) for all he wishes to say. His *articulation* of those words which he can speak is good, he has full power in the muscles of the face and the arm, and his hearing and vision are good. But there is a *defect in his mind* which prevents him from recalling the movements of articulation which indicate the word he wants to use. He knows the right word when he sees or hears it: hold up a pen and suggest to him that it is a haddock; he shakes his head and gets angry; write on paper or say "pen," and he nods and smiles. Such a case represents one of the commonest kinds of aphasia; it is a *loss of memory of the co-ordinated movements of speech*, and is known as **aphemia**.

Take another and rarer case, and suppose that instead of speech it is only the power of writing which the patient has lost. His motor power is good, his speech correct, and his vision and hearing perfect. But he cannot write a word, although he knows the word he wants to write. In short, he has *loss of memory for the co-ordinated movements of writing*, and the condition is **agraphia**.

Thus we have two kinds of possible defect of a patient's outgoing, initiating, or ideo-motor centres in the cortex corresponding to the two principal means by which he communicates his thoughts to the outer world (see table below).

IDEO-MOTOR APHASIA, *i.e.*, defect in the cortical mechanism of the centres of speech and writing.

	Tests.	Nature of Defect.	Position of lesions in cortex cerebri.[1]
Speech loss (aphemia).	Cannot talk correctly or say names of objects.	Loss of the educated recollection of the movements required for spoken words.	Posterior end of 3rd left (in most people) frontal convolution (Broca's) and lower end of ascending frontal (Fig. 158).
Writing loss (agraphia).	Cannot write names of objects or convey his thoughts in writing.	Loss of the educated recollection of the movements required for writing.	Posterior end of 2nd left (in most people) frontal convolution.

[1] Note that these lesions are ordinarily situated in the left hemisphere, but when the patient is left-handed they are situated in the right hemisphere.

(b) **Ideo-sensory aphasia** (sensory aphasia). Can the patient understand **written** or **spoken words**? Let us now consider the incoming forms of aphasia—visual aphasia (word-blindness) and auditory aphasia (word-deafness). They may be unattended by any defect of speech, vision, or hearing for ordinary purposes. But the patient has simply a loss of memory for the signs of other people's thoughts as expressed in written or spoken words.

First, as regards WORD-BLINDNESS (visual aphasia). We have a visual recording board (Figs. 157 and 158) in the cerebral cortex, upon which our recollection of printed or written words is recorded, so that after seeing them a few times, or being educated to read, we can recognise and name them at once when seen again. If the patient has *lost the faculty of understanding and recognising written or printed words*, the condition is word-blindness (visual aphasia). This form may be met with alone. The vision is good enough for ordinary purposes, but it is a curious circumstance that in most of such cases the patient cannot see with the left half of each retina (hemianopsia), because it so happens that lesions in the occipital lobe which produce word-blindness also cut off the optic radiations which come from one optic tract.

Secondly, as regards WORD-DEAFNESS (auditory aphasia), *i.e.*, the non-recognition of spoken words. As in vision, so in audition, we have a recording board in the cerebral cortex, by which we recognise spoken words. It is by the auditory memory that we learn to recognise such sounds as "mamma" and "papa," and afterwards other sounds in our own and other languages. When a patient's faculty of hearing for ordinary purposes is intact, but *he cannot understand spoken words*, the condition is word-deafness (auditory aphasia). This is the rarest form of aphasia, for the recognition of sounds and spoken language is the first thing we learn, and, as a general neurological principle, the faculties first to come are less easily damaged than later acquirements. It is practically always associated with word-blindness, and generally also with ideo-motor aphasia.

IDEO-SENSORY APHASIA, *i.e.*, defect in the cortical receptive centres for word-vision or word-hearing.

	Tests.	Nature of Defect.	Position of lesions in cortex cerebri.[1]
Word-blindness (visual aphasia).	Can see, but cannot read or recognise *printed* or *written* characters. Usually with left homonymous hemianopsia.	Loss of the educated visual memory for written (or printed) signs.	Angular gyrus (visual speech centre); or in the left occipital lobe, so situated as to sever the optic radiations between the angular gyrus and the visual centre.
Word-deafness (auditory aphasia).	Can hear, but cannot understand or recognise *spoken* words.	Loss of the educated auditory memory for speech.	Posterior half of the superior temporo-sphenoidal (Wernicke's) convolution (auditory word centre).

[1] Note that these lesions are ordinarily situated in the left hemisphere, but when the patient is left-handed they are situated in the right hemisphere.

I have described these four varieties of memory of the signs of thought singly in order to make the subject clear, but they are all interdependent one on another. The complete memory for a pen, for instance, involves a visual recollection of the object and its printed sign, an auditory memory of the sound "pen," and an ideo-motor recollection of the movements used for saying or writing "pen." In point of fact, the four varieties of aphasia which correspond to these forms of memory are nearly always mixed in various proportions, aphemia being the most common, agraphia the next, word-blindness the next, and word-deafness the rarest. This renders their investigation difficult. Par-aphasia was a term used by Kussmaul to indicate a slight degree of mental aphasia with varying admixtures of aphemia, word-blindness, and word-deafness, leading to the misplacing and misapplication of words. Par-agraphia was used by him to indicate slight degrees of mistakes in writing.

Lesions. Pure cases are, as just mentioned, very rarely met with, but now and then such cases have been observed, and we are thus able to locate the position of the several lesions in the cortex. These are given in the tables, and in Fig. 158. Usually the lesion involves the grey matter of the cortex, occasionally the white strands beneath The changes may be structural, as in cases of haemorrhage, embolism, thrombosis, or tumours; or functional, and leave no trace behind them.

Clinical Investigation. One so seldom meets with aphemia, agraphia, word-blindness, or word-deafness alone that the investigation of a case of aphasia is not easy, and it becomes a question as to which of these predominates.

(1) First ascertain whether there is any or how much defect in the articulation or motor apparatus of the face, tongue, or arm.

(2) You will find it convenient to proceed next to investigate the patient's power of comprehending your communications to him, in the inverse order of the description I have given above.

(3) Word-deafness, the rarest form of aphasia. Give the patient some simple spoken order, *e.g.*, "shut your eyes," but do not accompany your spoken orders by any gesture. If he responds, there is no word-deafness; if he does not respond he is either deaf (an unlikely thing on both sides) or he is word-deaf; and you must ascertain that he is not deaf by seeing if he turns his head towards a sudden noise or musical sound.

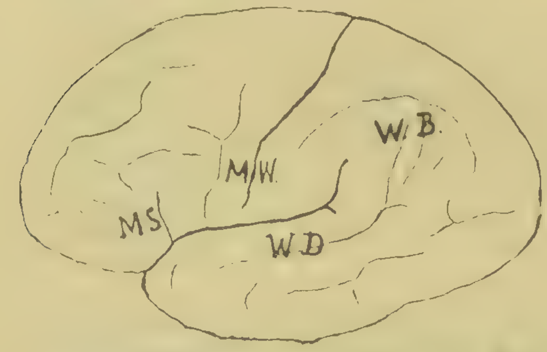

Fig. 158.—M.S., ideo-motor centre for speech. M.W., ideo-motor centre for writing. W.B., centre damaged in word-blindness. W.D., centre damaged in word-deafness.

(4) Word-blindness. Ascertain if his sight is good, and particularly if he appears to be able to see equally well with both halves of his retina, for word-blindness is commonly accompanied by homonymous hemianopsia. Give him the newspaper (upside down at first) and ask him to identify some of the letters. Then write out some simple instruction and show it to him; if his sight is good (except for hemianopsia) but he does not respond, there is visual aphasia (word-blindness). In some cases you can place a pen, a penknife and a watch before him, writing out their names on pieces of paper, and ask him to place them on the objects.

(5) Agraphia. Can he express his thoughts to you correctly in writing? Ask him to write his name, for instance, or write an account of his illness.

(6) Aphemia. Can he remember words he wants to say, or does he use them incorrectly in conversation? Can he name objects correctly? Hold up a pen and suggest wrong names for it, finally saying or writing "pen," and learn by his gestures if he knows which is the right name.

§ 529. Defects of memory (amnesia) may be (a) *entire*—forgetfulness for everything—or (b) *partial*—forgetfulness for certain facts.

(a) **Loss of memory as a whole** may be indicated by a forgetfulness for recent facts, such as what day of the week it is; or for remote events such as incidents of youth; or by a forgetfulness of what the patient wished to say or do (intention amnesia), or where he had placed things.

Causes. (1) *Neurasthenia* and various *debilitating conditions* such as anæmia, convalescence from severe illnesses, general ill-health, and exhaustion from overwork are commonly attended with some defect of memory. The activity of the memory, like that of the attention (or power of concentration of the thought), is indeed a measure of the activity of the mind, and when the brain is "tired" the memory flags. (2) The memory is also temporarily impaired under the use of certain *drugs*, *e.g.*, large doses of the bromides, but it readily recovers when this drug is stopped. (3) In *advancing years* the memory may become permanently deficient as a normal phenomenon (senile amnesia and senile dementia). The peculiarity of senile amnesia is that it applies chiefly to recent events and to recently acquired knowledge. The patient perhaps can give us full particulars of his early life, and repeat poetry learned in youth, but is unable to mention any event of the same or the previous day. (4) In the mental condition of *chronic alcoholism*, amnesia is a prominent and sometimes incurable condition. For instance, a lady who was under my care for alcoholic neuritis and delirium three years ago, has now recovered both in mind and body, except that she is unable to recall a single fact or incident for five minutes at a time. (5) Defective memory is also met with as a symptom of *dementia* and other forms of chronic insanity. The patient puts things away and forgets where they are; he also forgets where he is and what he was going to do. (6) *Sudden obliteration of memory* is not unknown. It may occur after a severe illness, and a portion or page of the recollection becomes blotted out, the patient picking up the thread of his life where it left off ten or twenty years before. The case is mentioned by Sir Wm. Gowers of a clergyman aged 60 who obstinately believed he was 40, and picked up the thread of his life at that age. Sometimes such sudden obliteration occurs after epilepsy or without known cause, as in cases reported in the papers from time to time of persons who have forgotten their name and all particulars about themselves.

(7) *Dual personality* (syn. "dual consciousness," "alternating consciousness") is another mental condition, in which loss of memory is a prominent feature. It is a rare and interesting condition, in which a patient lives alternately two different lives, or rather, has two alternating mental states which may for purposes of description be called state a. and state b. The essential peculiarity of the condition is that the patient when in state b. has no recollection of his thoughts and acts while in state a., and *vice versâ*. In each state he picks up the thread of his life when he was last in that state. Moreover, while in state b. he may behave in a totally different manner to that in which he behaved during state a. He is subject, in other words, to alternating states of consciousness and character, in which the whole mental attitude and mental record is changed. As a temporary condition dual consciousness may occur after epilepsy (masked epilepsy, or minor epilepsy chiefly) or after hysterical attacks. As a more permanently alternating condition it is found in some without this association.

(b) Among the **partial losses of memory**—

Mental aphasia may be mentioned. It is as we have just seen (§ 528), a loss of memory for the signs of thought.

Verbal amnesia is really a slight degree of ideo-motor aphasia manifested by an inability to recollect the words or names when the patient wishes to speak. It occurs with a tired brain or after some emotional shock.

Visual and auditory amnesia. Many interesting observations of late years go to show that the memory is compounded of different constituents. Apart from the association of ideas, there are two distinct means by which different individuals recall an idea. (i.) Some recall an idea by a visual impression such as the form or colour of the object it represents or by the image of the printed word representing it which they have seen or read. This is called a *visual memory*, and people who chiefly use this form remember those ideas best which were originally conveyed to them by pictures or objects seen, or by reading a descriptive passage to themselves. (ii.) Others can best recall an idea which is conveyed to them through their ears, *e.g.*, by sounds or by words read aloud to them, and these have what is called an *auditive memory.* Though everybody possesses both these forms of memory, they unconsciously make use more of one than the other. The visual memory of most people is better than their auditive memory, and they recall objects and pictures seen much more readily than sounds heard; hence the great value of kindergarten and demonstrative methods of education. Charcot narrates a remarkable case of a highly educated man, a banker, who, having an unusually good visual memory, suddenly, after a severe emotional shock, lost it completely. He was conscious of a great blank in his mind, and was unable to picture to himself any of the forms with which he had formerly been familiar, such as the shape of a building, a column of figures, the colour of his wife's hair, etc. By degrees, however, he learned to substitute his auditive memory, which hitherto had lain dormant, and so he gradually became able to carry on his business again.[1]

GROUP II. γ. *Acute perversions of the mind (delirium and mania).*

§ 530. Acute mental exaltation or excitement occurs clinically in two forms—Delirium and Mania—which differ, however, less in their clinical features than in the circumstances under which they occur. I. *Delirium* is the term applied to mental excitement which is clearly traceable to some bodily disorder of which it is a complication. II. *Mania* is the term applied to mental excitement when no such source can be traced, the mental condition being the only, or at any rate, the primary disorder. III. *Active* or *Acute* melancholia is occasionally met with; it only differs from chronic melancholia (§ 532) in the misery and depression of the patient being of a more aggressive character. Mania is practically always acute; melancholia nearly always chronic.

Clinical Investigation. The first and most important point in any given case of delirium or mental excitement to which you may be called for the first time is to ascertain the temperature, for the most useful clinical division of the causes of delirium is into *Febrile* and *Non-febrile.* Secondly, it is important to make a thorough and complete investigation of all the organs of the body, to ascertain whether there be any local inflammatory disorder, such as pneumonia, with which delirium may be connected, either directly or indirectly. I remember once overlooking a case of latent pneumonia and consigning it to the lunatic ward of the workhouse. The urine also should be carefully examined for albumin, sugar, or other abnormality. Thirdly, an inquiry should be made into the history of the malady, and of the patient, especially as regards alcohol. In reference to the etiology of delirium, three important *predisposing causes* have to be borne in mind. First, there is a marked predisposition in some nervous people to develop delirium in presence of a slighter cause than would be operative in others. Secondly, there is a marked hereditary tendency towards the same vulnerability; and thirdly, excessive drinking predisposes to the occurrence of delirium after an injury, operation, and many diseases which are not usually so attended.

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[1] "Leçons Cliniques sur les Maladies du Système Nerveux," Tome iii., Leçon 13.

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I. The causes of **delirium** have been considered (p. 576) and need only be here enumerated.

Febrile.	*Non-febrile.*
Diseases of the brain—especially meningitis.	Delirium tremens.
Acute visceral inflammations.	Chronic renal disease.
Acute specific fevers.	Post-febrile delirium.
Delirium tremens (rare cases).	Reflex delirium. Deliriant drugs.

II. **Acute mania** may supervene suddenly—(1) during convalescence from exhausting diseases (as previously mentioned); (2) in the course of other diseases of the nervous system (*e.g.*, G. P. I.); (3) in the course of some other form of insanity. Its onset is usually rapid, tongue-tremor being often met with in the early stage (Dr. G. H. Savage). The stage of excitement is soon reached—loquaciousness, sleeplessness, continual restlessness, incoherence in which delusions and ideas succeed each other with great rapidity, sometimes relating to moral and religious, at other times to intellectual topics. After lasting some weeks or months, recovery (sometimes quite suddenly) ensues; sometimes it is followed by moral or mental obliquity, or dementia; rarely, it passes into chronic mania. The temperature is normal throughout. In most cases there is a tendency to relapse.

Acute delirious mania (Bell's Mania) is an acute maniacal condition coming on suddenly in a person in apparent health, attended by pyrexia, usually running a rapidly fatal course, no lesions being found after death. It is happily a somewhat rare disease. The symptoms come on abruptly, and quickly amount to frenzy, accompanied by outbreaks of great violence. The temperature ranges irregularly from 100° to 104°, and in the course of one to three weeks the disease terminates in great bodily prostration and usually in death. Acute delirious mania differs from acute mania in the elevation of temperature, the rapid wasting, and its more rapid and fatal termination. It resembles some cases of enteric fever very closely, acute pneumonia and acute meningitis, but their proper symptoms are absent.

The *treatment* of mania consists mainly in the administration of food (with stimulants if the pulse so indicates). Narcotics and depressants may be tried, and Dr. Savage mentions a case which apparently got well under frequent small doses of opium. For acute delirious mania the wet pack or the graduated bath (p. 667) may be given. Professor Osler recommends venesection.

III. **Acute dementia** (**"stupor"**) is a rare condition of sudden ablation of the mental faculties.

GROUP II. δ. *Chronic perversions of the mind (insanity).*

§ **531.** This is a more complex group than any of the preceding, and it forms the collection of morbid conditions of the mind, known as insanity. Insanity may be defined as a serious departure from normal of one or more of the various mental processes (the senses, the intellect, the emotion and the will) which control the conduct of the individual, and thus render him liable to be dangerous to himself or others. The subject may be briefly dealt with under—

Chronic Mania;
Chronic Melancholia;
Chronic Dementia;
Various Special Types of Insanity; such as general paralysis of the insane; delusional insanity; hysterical or emotional insanity; moral insanity; alcoholic, syphilitic, and puerperal insanity; catalepsy; trance; and lastly,
The mental defects in childhood and adolescence.

Clinical Investigation. I have already, in § 512, referred to the importance of tact and general knowledge in investigating psychical disorders, and I may here mention two other points: (1) get your data in chronological sequence as far as possible, and (2) never be in a hurry. Any careless, inept, insistent, or rapid questioning will only confuse and silence your patient and defeat your object. Gain his confidence; let *him talk to you* first of his favourite hobby, then of his thoughts and feelings, and finally of his delusions or hallucinations. The main points to investigate in mental cases are sleep (and dreams); speech; writing; memory; decision and will; reasoning power; moral and ethical standards; delusions; hallucinations; and lastly, whether the patient regulates his conduct according to these, *i.e.*, whether he (or she) is a potential danger to himself or others.

The three terms *delusion*, *illusion*, and *hallucination*, strictly speaking, have different meanings, but they are used somewhat laxly and indifferently. A hallucination is a false sense impression without any external stimulus, *e.g.*, the hearing of voices and the seeing of snakes. *Illusions* are distortions of external stimuli, as in alcoholic delirium, when a lady's muff is thought to be a cat. *Delusions* are false ideas existing in the mind of a patient, as when he believes himself to be the Deity. Hallucinations are met with particularly in mania, delirium, and mania *a potu*, and it is surprising what minute details can be given to us about these creations of the mind. Hallucinations of sight (rats and snakes) are much less common than those of hearing (hearing voices). Hallucinations of taste and smell are present not infrequently in association with delusions of being poisoned. Hallucinations of common sensation are both frequent and various, and are especially frequent at the climacteric, and in hysterical subjects who become insane. Amongst the varieties of sensation, hallucinations of the male or female sexual organs are not infrequent, and in this way false accusations may be made against those (especially doctors and nurses) with whom the patient comes in contact.

In regard to all delusions, illusions or hallucinations, it is important to estimate as far as possible how far such perversions of the mind influence, or are likely to influence, the acts or conduct of the individual.

After noting the age of the patient, the first question to investigate in the history is whether the attack came on with excitement or with depression. If with excitement, the case may be one of mania or general paralysis of the insane or delirium (§ 530). If the case came on with depression, it may be melancholia or G. P. I. If the patient is an adult, start at § 532; if an adolescent at § 538; if a child under 10, start at § 539.

§ 532. I. **Chronic Mania** is simply a prolonged form of acute mania as previously mentioned (§ 530), lasting with less excitement for years, instead of for weeks or months. Recurrent mania is that which recurs, sometimes at the menstrual epoch. "Folie circulaire" is an alternation of mania and melancholia, with lucid intervals. Monomania was used by Esquirol to indicate a form of delusional insanity (§ 535) dominated by one fixed idea; it is not really mania.

II. **Chronic Melancholia** is a morbid condition of miserable self-consciousness and self-negation without hope. Melancholia occurs under three circumstances: (1) it may be part of some other mental disorder, such as G. P. I., or a stage (first or third) of mania; (2) it may constitute the whole of the mental disease without previous ill-health; or (3) it may supervene on neurasthenia or some bodily ailment. The onset is usually insidious, and commences with extreme self-consciousness, combined with sadness, as indicated by tears without cause, and when the patient is remonstrated with he is irritable. There are morbid dreads of impending calamity which cannot be named, sleepless nights, and a suicidal tendency. Among the physical signs commonly noted in such cases are feeble circulation, as evidenced by cold feet and chilblains, and constipation. Melancholia differs from hypochondriasis in the "hopelessness" of the former and apathy to surrounding conditions.

Four *varieties of melancholia* are described—active, passive, and simple melancholia, and melancholic stupor. (1) and (2) *Active* and *passive* melancholia depend upon the degree to which patients give expression to their grief. In the former, they are always imparting their trouble to some one; it is in reality an acute melancholia. In the latter they sit for hours together in a dejected state, and it is sometimes difficult to elicit their leading delusion. (3) *Simple* melancholia may be described as melancholia without definite delusions. It consists simply of misery, sleeplessness, self-blame, and inability to continue at work. This form is common in the over-worked or much-worried, and in women at the climacteric. Suicide is not uncommon in these cases, and precautions, which are sometimes neglected on account of the simplicity of the affection, should not be omitted. Otherwise the prognosis is favourable. (4) In melancholia with stupor (melancholic stupor) the patients remain speechless and motionless, with an aspect of abject misery. Their limbs may be flaccid, or in cataleptic rigidity. They are abstracted, and oblivious to all external stimuli. They resist external interference, but are not usually violent. The condition may come on suddenly or gradually, and last for months or years. Some cases end fatally in a short time. Some have recurrent periods of exaltation. Some terminate in a condition of permanent weakmindedness, and a few recover. It is equally common in both sexes, but is more frequent in the young than the old. Sometimes it follows a severe and exhausting illness; and sometimes it follows acute mania.

Course and prognosis. The melancholic process is longer than the maniacal one. The duration varies considerably, but lasts an average of some three to twelve months. Relapses are not infrequent. The slower the advent of the disease, the slower is the recovery. Recovery is common enough in the young, the prognosis being worse as age advances. Suicide is frequent in all forms, but death from the disease is rare. Heredity is an important factor, and the nutrition of the body at the time is another. The melancholia of pregnancy is favourable, but the melancholia of lactation is always grave. It is interesting to remember that cases of melancholia may recover even after a very long time (9, 10, and 13 years have been recorded). There is a distinct *suicidal tendency* in all cases of melancholia; but those are specially liable, according to Dr. Savage, who have delusions of impotence, of being followed or persecuted, of hearing voices, of being the cause of injury to their relatives, or who are suffering from great physical weakness or bodily disease. The tendency to suicide appears to increase with age, and suicidal cases generally have an insane heredity, and often a suicidal heredity also. Patients generally have a special predilection for some particular mode of death, one to poisoning, another to drowning, another to hanging, and another to blowing his brains out, and they will often avoid other means which may happen to present themselves.

In regard to *causation*, melancholia depends in most cases more upon physical and external, than upon mental and moral causes. Some of the commonest causes have been referred to under the variety simple melancholia. No doubt want of society, solitary habits, combined with a sedentary life, in which the person is debarred from genial companionship, are the prominent causes of this condition. A general depression of the vital powers, *e.g.*, from bodily disease, fevers, heart disease, etc., is an important factor. Any age may be affected, but it mostly arises at or after middle life.

Treatment. In the simpler cases, such as those referred to under simple melancholia, a few weeks' rest under supervision, with a pleasant companion, and complete absence of the conditions under which the disease arose, followed by a few months' easy travel, will generally set the patient right. If the interest can be aroused, and the attention attracted for a sufficient time, much can be done to relieve the condition, and even remove the delusions. Feeding is necessary, and in case of refusal it may be done by means of (a) a spoon, pouring the fluid into the cheek beside the teeth, or (b) by the nasal or stomach tube. The quantity thus administered should be equal to 5 pints of milk, 2 pints of strong beef-tea, 6 eggs, and 3 to 6 ounces of brandy per diem. Suicide must be prevented by removal to an asylum, or careful watching at home.

§ **533.** III. **Dementia** is deficiency of all the mental faculties, coming on in adult life. It comes on as a *primary* condition in (a) chronic alcoholism, and (b) advanced life (senile dementia). The first, as we have seen, shows itself especially by a loss of memory. The second also has the same peculiarity, with the additional feature that the memory is lost for recent events only. Dementia comes on as a *secondary* condition in (a) general paralysis of the insane, and as the concluding stage in many other forms of mental disease; and (b) after vascular and other gross intracranial lesions. Even after a small lesion of the brain the mental capacity for business is hardly ever as good as before its occurrence, and the patient often becomes childish, peevish, forgetful, emotional, and by degrees in severe cases, completely demented.

The **special forms of insanity** are—general paralysis of the insane; delusional insanity; emotional insanity; moral insanity; alcoholic, syphilitic, and puerperal insanity; catalepsy and trance.

§ **534.** IV. **General Paralysis of the Insane** (G. P. I.: Paralytic Dementia) is a progressive generalised muscular weakness and tremor, accompanied by mental symptoms, often of a grandiose character, occurring almost entirely in men in the prime of life; due to atrophy and a scattered sclerosis of the cortex cerebri. This change represents a later stage of some nutritional alteration in the nerve cells: Dr. W. F. Mott has shown that the changes in the cerebral cells (swelling) produced by ligature of the cerebral arteries, and so cutting off the nutrition, resemble those found in the early stages of G. P. I. The disease depends on a parasyphilitic process, *i.e.*, it is an indirect or nutritional effect of the syphilitic toxin (resembling tabes dorsalis in this respect), not a direct manifestation of syphilitic lesions.

Symptoms. Paralysis of the limbs may sometimes exist for many years without mental symptoms (*vide infra*). In my own experience, mental have generally preceded the physical symptoms, but this order varies. The characteristic symptoms are mental alteration, general weakness, tremor, and alterations in the pupils and the speech. They are divided for convenience into 3 stages, each of which lasts about one year. In the first or *premonitory stage* (the stage of irritability and muscular tremor) irritability, restlessness, perversion of the moral sense, and loss of the faculty of attention are among the most usual features, though a variety of other mental aberrations are met with. A man of even temper, who has been a fond husband and father, becomes irritable over trifles, gives way to coarse and blasphemous language, exhibits sexual aberrations, or commits theft. He becomes egotistical, showing the delusions of grandeur so characteristic of the malady, and squanders his money. He may believe himself to be very strong, very wealthy, or very high born. Sometimes, on the other hand, great depression is the characteristic of this stage; and thus, even in this period, we find two groups, one with a tendency to expansion and restlessness, the other with a tendency to despondency and loss of energy. Accompanying, preceding, or following the mental symptoms are various *physical changes*, amongst the commonest of which are (i.) tremor (fine, small, and rhythmical) of the hands (giving rise to characteristic handwriting), and of the lips and tongue (giving rise to a very characteristic speech, viz., a slurring of the words as in intoxication). (ii.) The pupils in this stage are usually small, very contracted and immobile to light ("pin-point pupils"); very often unequal. (iii.) Headache, neuralgia, and various subjective sensations are complained of. (iv.) Weakness of the limbs, always generalised, and sometimes combined with some inco-ordination. As a rule, the knee-jerks are increased in G. P. I. Sometimes the symptoms of lateral sclerosis are present. Anæsthesia and other alterations of sensation and of the special senses are frequent. The *second stage* (stage of fits) is characterised by (i.) mental enfeeblement which replaces the exaltation in the first stage; (ii.) increasing muscular weakness, difficulty in walking any distance, and especially in the act of turning, sometimes combined with giddiness. (iii.) Fits are almost invariably present at some time during this stage; they vary in character, but are usually syncopal or epileptiform, with or without the loss of consciousness. Sometimes they consist of attacks of numbness of the limbs, or aphasia, or coma. The *third stage* is the stage of progressive mental extinction. The speech becomes inarticulate, the paralysis extreme, and may be accompanied by contracture, so that the patient cannot feed himself. His mind undergoes progressive extinction, and there is loss of all its faculties. The urine and fæces are passed involuntarily.

Many different *varieties* have been described, but these only exist in the earlier stages of the disease; they all tend to one common form of progressive mental enfeeblement. (1) The expansive variety is the commonest, and forms the basis of the above description. (2) The melancholic variety is characterised by great depression and melancholic delusions. (3) In the paralytic variety, paralysis and tremor predominate with few or no mental symptoms, excepting occasional outbursts of emotion and some change in character. Sometimes the paralysis predominates in the legs (ascending variety): the legs may be rigid (spastic form, Savage); and there is an ataxic form closely resembling tabes dorsalis at the outset. (4) A congestive variety has been described, chiefly characterised by fits of various kinds.

Course and prognosis. The duration varies widely from a few months to three or more years, and the proportion occupied by these various stages differs greatly. One thing is very characteristic of the disease—remarkable intermissions of comparative or complete return to health. But these cases always break down on attempting to resume their former state of life. The expansive form above described usually runs its course in about three years. Where depression and melancholia are marked features, the prognosis is worse (Bristowe), and the disease is more rapidly fatal. The spinal paralytic or ascending form occupies a much longer period, and may extend to six or eight years. When the malady is once established, it invariably progresses towards a fatal termination.

This disease, on account of its great variety, has to be *diagnosed* from many different complaints, of which the following is a summary—(a) from other forms of *mental disorder*, chiefly by the paralytic symptoms, tremor, speech, and pupillary changes; (b) from other diseases giving rise to *generalised paralysis* (see § 568); (c) maladies attended by tremors and other neuro-muscular symptoms, such as *disseminated sclerosis* and *paralysis agitans*. Chronic *alcoholism* and *peripheral neuritis* are difficult to differentiate sometimes; they are recognised by a history or evidence of alcoholic dyspepsia, by absent knee-jerks and comparative absence of mental symptoms, excepting loss of memory. *Bulbar paralysis* is recognised chiefly by the symptoms being confined to the mouth, tongue, and throat, the mind being usually normal. The diagnosis from *tabes dorsalis* is not usually difficult, but these two diseases are very apt to occur in a mixed form.

Causation. (1) Adult males, in the very prime of their strength and manhood, that is, between 30 and 40, are the favourite subjects of the disease, but it may occur at any age. The youngest I have known was 16, the oldest 60 (an elderly man who married a young wife). It is generally said to be four times as common in men, but I believe it to be commoner. It is more frequent in the lower classes of life. A neurotic heredity is said to be in operation in as much as 30 per cent. of the cases. There seems but little doubt, however, that the disease is in its essence a syphilitic, or rather parasyphilitic process as above mentioned. Alcoholic, sexual, and other excesses, anxiety, and mental fatigue, are accessory causes.

Treatment. Something may be done in the way of *prevention* when there is a history of heredity by the avoidance of overstrain and of alcohol. Patients with a hereditary taint and premonitory symptoms such as the above should certainly avoid matrimony. The most important *remedial* measure consists of the removal of the patient from the conditions under which the disease has arisen, and especially avoidance of business and all causes of anxiety or mental strain. He should live a regular life, with outdoor exercise and amusements which take him out of himself. Iodides and nerve tonics (especially cod-liver oil) sometimes do good in the earlier stages. When excitement is present the head should be kept cool (possibly with an icebag) and the feet warm, and the bowels should be freely opened. Physostigma or hyoscyamin may be given; and if much excitement is still present, 30 ♏ tinct. digitalis every four hours, or a warm bath followed by an icebag. The cold pack is also useful, and bromide and sulphonal. In the melancholic varieties, arsenic, iron, and quinine are the only remedies that have been found useful. Galvanism to the central nervous system has not, so far, been attended with much success.

§ 535. **Other special types** of insanity are named according to their clinical features, such as delusional, hysterical and moral insanity; or according to their etiology, such as alcoholic, syphilitic and puerperal insanity.

V. **Delusional Insanity** is a chronic form of insanity in which the leading or sole mental alteration consists of a fixed delusion or hallucination, which modifies the conduct of the individual (compare clinical investigation, § 531) A delusion, illusion, or hallucination may arise under 3 conditions—(i.) it may arise in a person who is otherwise perfectly sane; (ii.) it may be associated with other evidences of insanity; or (iii.) it may, when no other symptom is present, constitute in itself delusional insanity, when, that is to say, it controls the conduct of the individual. Delusions, especially on religious subjects, are not at all uncommon in the so-called sane. But when these delusions modify the acts or conduct of the individual and lead him to act in an unusual manner, the condition becomes one of insanity. Patients who believe they are persecuted or followed are apt to be dangerous and either homi- or suicidal. **Paranoia** is a term which has long been in use in Germany, and has gradually been introduced into England. My conception of the term is that it is almost identical with delusional insanity but with a tendency to hallucinations. It does not always amount to insanity, since it may be applied to the eccentric personages who have some marked and permanent delusion, but who are otherwise sane. Such subjects are, however, responsible for many crimes. Voice hearers and those with delusions of love or persecution are the most dangerous. **Monomania** is a term, originally used by Esquirol, which has now almost fallen into disuse. It indicates, strictly speaking, a mental state which is dominated by one fixed idea or delusion—usually of self-importance—and in this sense it might be a symptom of G. P. I. Later on its significance has become more general, and it has been used by most to indicate a chronic form of insanity (which may follow an acute attack) in which the patient, though fairly sane on most subjects, has some one intellectual peculiarity or delusion. Many such persons are at large and pass for sane people.

VI. **Hysterical** or **Emotional Insanity**. The mental perversions to which hysterical subjects are occasionally liable are (1) emotional insanity, (2) hystero-epilepsy with insanity, (3) ecstasy, (4) catalepsy, and (5) trance. The first is an emotional mania often of a religious kind—a religious veneration for the curate, for instance. The patients are rarely or never suicidal or melancholic, though they may be passionate, mendacious, mischievous, crafty, noisy (screaming and singing hymns), and given to various kinds of movement such as hammering and dancing, or to the striking of attitudes (as in ecstasy). Perversion of the tastes and the appetite and a general capriciousness are very characteristic.

Cases of the class under consideration are relatively frequent, and in general terms, the *prognosis* of such cases is more favourable than many other forms of insanity, especially if no hereditary mental taint exists, and due skill and judgment is employed in their treatment. In the *treatment* of these hysterical mental affections, we should, as far as possible, avoid putting them to bed, for complete want of will, that is, lack of energy and initiative is a characteristic feature, and they will soon become bedridden. Change of environments, interests in life, and judicious companions are the central points. It is seldom necessary to send them to an asylum. Artificial feeding may be required, but should be discontinued as soon as possible. Savage suggests adding salt to create an artificial thirst, and then placing fluid nourishment in the way of the patient when, as sometimes happens, she is too lazy to feed herself. Laziness leads to dirty habits and the passing of fæces in the bed. We should look out for tubercle in these cases; organic disease of the brain has sometimes been found.

VII. **Moral Insanity** is recognised by some as a special form of insanity, in which the mental disorder consists principally, and sometimes solely, of a marked deflection from the normal standard of morality. The intellect and the will may be normal, and the emotions under control. In the adult, moral and ethical perversions occur (1) very frequently as an *early phase* of G. P. I., and many other forms of insanity, for, as Esquirol remarked, moral alienation is but the "first step to madness." (2) It is also met with in the adult *after recovery* from an attack of mania or other mental disorder. The patient, especially if young, is frequently left with a sort of moral scar, and the lower or animal side becomes prominent. (3) Occasionally one finds in adults a moral defect as a *substantive condition* without previous insanity and without any other mental defect. But often the childhood of such patients presented some similar defect in a less degree, for the condition is more frequently hereditary and congenital. *Kleptomania* (in which the patient is afflicted with an irresistible impulse to thieve) belongs to this class. Other patients have irresistible erotic or amorous tendencies, and others have a tendency to unnecessary exaggeration and lying. Dipsomania is a paroxysmal irresistible craving for drink. (4) *Moral insanity* as a substantive disorder is chiefly met with *in children*, particularly those of alcoholic, insane, or epileptic parents. In early childhood they may be perverse, mischievous, cruel, untruthful, or thieving. They are often precocious, and they may even be intellectually gifted. Nevertheless, such children generally need incarceration in course of time, if they do not find their way into prison. It seems probable that a large proportion of the criminal convictions among the children of the lower orders are subjects of this malady. The condition, as a rule, is incurable, unless by educational and disciplinary measures.

VIII. **Alcoholic Insanity.** Alcohol is generally accorded the chief place in the causation of insanity. It may result in two special forms—(a) delirium tremens, and (b) alcoholic dementia; it may also result in (c) delusional and other forms of insanity.

IX. **Syphilitic Insanity.** (a) After contracting this loathsome disease it will prey on the minds of some persons to such an extent as to produce an extreme degree of hypochondriasis, to which the term *syphilophobia* is aptly applied. In every symptom, normal or abnormal, they see the disease. (b) Syphilitic endarteritis may lead to a *dementia* indistinguishable from senile dementia except by the age of the patient. Various forms of mania and melancholia may also be associated with arterial and gummatous lesions, or with concurrent cachexia. (c) General paralysis of the insane is a parasyphilitic process (§ 534).

X. **Puerperal Insanity** is a generic term for the mental disorder which arises under three different conditions. (a) The mental perversion which arises during *pregnancy* is always a form of *melancholia*, and varies in degree from a simple exaggeration of the morbid longings and perverted tastes which are more or less usual during gestation to melancholia of a pronounced type, accompanied, perhaps, by delusions and a suicidal tendency. It is not generally serious unless heredity is in operation, and usually disappears after the confinement. (b) Puerperal *Mania* (or Puerperal Insanity proper) arises usually between the first and fourth weeks after delivery, coming on usually suddenly with maniacal symptoms. (c) The Insanity of *Lactation*, or post-puerperal insanity, is a form of *melancholia* which arises during the first two or three months, or any time during the first twelve months after confinement. All three forms are apt to recur in succeeding pregnancies, but unless there is a hereditary taint of insanity, the prognosis is good for recovery. Abundant food and removal from home are indicated, and special measures should be directed to meet the tendency to suicide or infanticide which is frequently present even in the mildest cases, and gives no indication of its presence until some untoward event occurs.

XI. **Catalepsy** (καταλεψις, a seizure or attack) may be defined as a state in which the patient is deprived of sensation and voluntary motion, in which the limbs remain in any position in which they are put. The patient may appear, but it does not follow that she is, unconscious to the surroundings. The eyes may be open, but she appears totally oblivious to all the outside world, and she may lie for hours, or days perhaps, passing her motions under her. These patients are nearly always of the female sex, and are invariably the subjects of some other hysterical manifestations, to which category, indeed, the condition belongs. Sometimes these cataleptic attacks are ushered in by hysterical convulsions, or a hysterical faint. They are usually determined by a fright or some emotional storm.

XII. **Trance** is a condition allied to catalepsy, in which the limbs are either rigid, or, more usually, flaccid, and lacking the feature of remaining in any one position in which they are placed. The patient may remain for weeks or months in what seems to be a faint, taking no notice, eating no food, making no movement, and scarcely breathing; though here again she may not be as unconscious as she seems. The pulse is hardly perceptible at the wrist, and unless the patient is forcibly fed she may ultimately die, but it is surprising the length of time she may live with hardly any nourishment.

§ 536. **Prognosis** and **treatment** of insanity in general. The *course* and *prognosis* in several of the various forms of insanity have been referred to. In general terms the chief points on which the prospect of recovery depends, are (1) the absence of heredity, especially direct heredity; (2) the rate of onset of the attack, being more favourable in a rapid than a slow insidious advent; (3) the duration of the attack before the patient comes under treatment; and (4) the kind of insanity present.

PROSPECT OF RECOVERY IN CHRONIC MENTAL DISORDERS.

Good.	*Moderate.*	*Bad.*
Hysteria and emotional.	Delusional.	G. P. I.
Alcoholic.	Mania.	Moral insanity.
Syphilitic (mostly).	Melancholia.	Dementia.
Puerperal.		

The *treatment of insanity* in detail has been referred to under the different forms, but the general principles resolve themselves into three indications—(1) feeding; (2) change of environment; and (3) placing under restraint. Hypnotism is available for some of the slighter cases, especially where alcoholism is in question.

The question of **removal to an asylum** depends on many things, chiefly (i.) the manageability of the patient, (ii.) the means at home for control, and (iii.) the character of the mental disorder and its potentiality for homicide or suicide. The procedure for removal is somewhat intricate, and it is useful to remember that the relieving officer of the parish is a most convenient person to apply to, bearing in mind, however, that it is no part of his duty to undertake private cases, but that nevertheless if he be approached with due regard to the importance of his office, he may save those concerned a great deal of trouble and supply them with all the necessary forms and particulars as to modes of procedure. He is also in constant relation with the lunacy justices.

Procedure for removal of lunatics and **alleged lunatics**. Any person either pauper or non-pauper *deemed* to be a person of unsound mind can for his own safety or that of the public be removed by a policeman or relieving officer to the workhouse and can be detained there for 3 days upon the certificate of the constable or relieving officer, and upon the certificate of the Medical Officer of the Workhouse for a total of 14 days. Meantime the procedure under No. 4 below can be instituted. This method is now often utilised for persons in all classes of life who are dangerous and away from their friends.

Similarly, a person deemed to be a lunatic and wandering at large not under proper care can be apprehended by a constable, relieving officer or overseer of the parish and taken to the workhouse.

In *private cases* the urgency order (1 below) can be used in urgent cases; this holds good for 3 days from date of signature; if not urgent (2) is the usual method.

All the different forms necessary are procurable from Shaw, Fetter Lane, London, or as previously mentioned, the relieving officer often has some at hand.

A patient can be removed to an asylum in England or Wales in 5 ways.

(1) Under an Urgency order signed by a relation (or guardian), and one doctor.
(2) Under a Reception order of a Justice obtained by petition of relative on two doctors' certificates.
(3) Under an order after Inquisition, being a written authority from the "Committee" of the person, together with an office copy of the order of the Court of Chancery appointing the "Committee." The "committee" is a legal phrase for the guardian appointed by the court.
(4) Under a Summary Reception order of a Justice,
  (a) on information from the police or relieving officer that a non-pauper is cruelly treated or neglected, a justice calls in two doctors who certify insanity;
  (b) on information from the police or relieving officer that any person, pauper or not, is wandering at large deemed to be a lunatic, a justice calls in one doctor who certifies insanity;
  (c) on information from the relieving officer that a pauper is deemed to be a lunatic, a justice calls in one doctor who certifies insanity.
(5) Under a reception order by two Commissioners (rarely done) who call in a doctor who certifies insanity.

Idiots and imbeciles from early life can be removed to an idiot or imbecile asylum on one medical certificate and a statement by a relative.

The procedure in Ireland and Scotland is somewhat different.

Testamentary capacity. The intricate technicalities of the lunacy law cannot be entered upon here, but a knowledge of what constitutes the testamentary capacity of a patient is of great importance to the practitioner because it is often on his evidence that courts of justice decide such matters. The testamentary capacity of a person depends practically on three questions—

1. Did he at the time understand the nature of the will and its effects and the extent of the property of which he was disposing?
2. Did he provide for his relatives, or if not why did he leave them out?
3. Had he any delusion bearing on testamentary matters?

If these questions can be satisfactorily answered and proven the will is valid however eccentric the patient may have been on other points or even if he was at the time a certified lunatic.

§ 537. **Hypnosis** may be defined as a condition resembling sleep in which the subject's capability to receive and act upon suggestions is greatly increased. This increased suggestibility is made use of by the operator for the implanting of new and healthy conceptions and the removal of morbid ideas, the object being to influence the body through the mind. It is worthy of study both from a psychological and a medical point of view. It explains many of the cures performed at Lourdes and other shrines, and by faith-healers in all parts of the world.

Bernheim asserts that 80 per cent. of his hospital patients are hypnotisable to the extent of somnambulism, characterised by amnesia on waking;

in his private practice the proportion was much less. Dr. Lloyd Tuckey and the author, in the course of some experiments at Paddington Infirmary, estimated that only about 5 per cent. of the patients there were hypnotisable to that degree. Anæmic young women are perhaps the most easily hypnotised, but strong and healthy men are often susceptible, and I have often found other people in whom there is not the faintest suspicion of hysteria are subject to hypnotic influence. Wingfield found the undergraduates at Cambridge particularly good subjects, and soldiers and others accustomed to respond to the word of command are very amenable to hypnotic suggestion. Children, too, are good subjects when able to understand what is expected of them, whereas the insane and imbecile are generally unhypnotisable, and hysterical women are by no means the best subjects. It is only by trial one can determine whether a person is hypnotisable.

There are various *methods of hypnotising* which are easily acquired. The patient should be put at his ease and seated in a comfortable chair. He is made to relax all his muscles and to fix his gaze on a bright object held about twelve inches above the eyes so as to cause a slight strain and convergent strabismus. This is the method used by Braid, and most susceptible persons will fall into a cataleptic or somnambulic condition when submitted to it without further procedure. But hypnosis is often assisted by verbal suggestion of sleepy sensations, such as heaviness of the eyelids and limbs and increasing torpor of mind and body. "Passes," as the mesmerists called them, made by passing the hands, fingers extended, about half an inch from the face so as to create a slight draught of air are often helpful in producing and deepening hypnosis. Whatever method is used the *rationale* is the same—it consists of monotonous stimulation of one or more senses with corresponding inhibition of others, leading to a condition of altered consciousness in which organic functions as well as mental states become more under the control of the will. The deeper the hypnosis the greater as a rule the effect of suggestion, but good therapeutic results are obtainable when only a slight drowsiness is produced. The patient should be allowed to rest quietly for half an hour, and should be told to awake without shock or feeling of discomfort at the expiration of that time either spontaneously or on a given signal. The suggestions should be given in an authoritative and impressive manner, and may be accompanied by manipulation of the affected part as practised by Braid. They are directed towards the removal of pain, spasm, and other symptoms, and the re-establishment of normal functions. Such suggestions often act immediately, and the action in successful cases is continuous and sustained.

Hypnotism should be used only by medical men and with proper *precautions*. The consent of the patient and his friends should be obtained, and a third person should be present during the operation. In competent hands no bad effects result from its employment even over prolonged periods, but much evil, moral and physical, might follow the abuse or misuse of this powerful agent. Its use for purposes of public exhibition should be forbidden by law.

Uses. Therapeutically, hypnotism has been employed to relieve pain, to procure sleep and rest, to remove delusions and obsessent ideas, *e.g.*, agoraphobia, to cure the effects of grief and shock, to reform alcoholics and moral perverts, to cure various neuroses such as nocturnal enuresis, writer's cramp, to relieve various hysterical manifestations such as anæsthesia, attacks, and paralysis. Witterstrand of Stockholm claims to have cured many cases of epilepsy by keeping patients in a state of profound hypnosis for three weeks continuously. In exceptional cases hypnotism has been employed as an anæsthetic in surgical and midwifery practice.[1]

§ 538. Mental deficiency in adolescence (10 to 20 years) may (1) consist occasionally of one of the chronic mental disorders of adults previously described, and especially the MORAL INSANITY described on p. 961; or (2) it may be a SEQUENCE of either of the two groups below (congenital and non-congenital mental deficiency of childhood) see § 539. (3) There is also a PRIMARY MENTAL DEFICIENCY inherent in the individual and manifested for the first time in adolescence, and it is this last class with which we are now concerned; it includes the "borderland cases," and constitutes at once the puzzle of the alienist and the trial and burden of relatives and guardians.

Symptoms. Various kinds and degrees of defect may be met with. It is nearly always between the ages of 10 and 20 that such cases come under notice for the first time, sometimes because "they will do stupid things," sometimes for "romancing" or lying, sometimes because they do not learn as rapidly as others, or "are not so bright," sometimes because they are unruly or have taken to drink or other vices. In the last-named case, when they have a moral obliquity, they may be possessed of brilliant intellectual gifts, but more often there is an all-round deficiency and they are and remain childish all their lives. This deficiency leads them to consort with all sorts and conditions of men and women, whose habits and language they quickly imitate; if sent for a sea voyage, they are generally to be found in the forecastle or stewards' pantry, and rapidly assume the indelicate language of the seafaring man. Their parents expostulate with them, and they promise, and do try, to avoid repeating the offences, but they soon break out again. Much trouble accrues to the parents or guardians of such persons to keep them when belonging to the well-to-do classes of life from squandering a fortune, forming an ill-judged liaison, or getting into other and worse troubles, and when belonging to the lower classes, to keep them out of prison.

Causes. In nearly all such cases there is a neuropathic family history on one or both sides. A few cases can be traced to some of the causes of acquired mental deficiency previously mentioned. Inbreeding such as occurs in the aristocracy may be a contributory cause. The condition would naturally be aggravated by faulty education and mode of life, but without the hereditary and inherent mental defect it cannot be produced.

The *treatment* is a question of careful training and education, and a good deal can be accomplished if no expense is spared. It is always best,

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[1] Those who wish to study the subject of hypnotism more closely should consult "Psychotherapeutics, or Treatment by Hypnotism and Suggestion," by Dr. Lloyd Tuckey, 4th edit., London, 1900, Baillière, Tindall & Cox; "Hypnotism, Its History, Practice and Theory," by Dr. Milne Bramwell, or the classical works of Charcot and Bernheim.

I am sure, to remove these cases from home, for nearly always a state of friction arises between the girl or youth and one or other parent, which is detrimental to their progress. If the patient is liable to outbursts of passion these must be gently curbed, and the system of education made attractive by utilising any particular taste which they have. The amount of improvement which such cases are capable of depends upon the possibility of fixing the Attention, and this depends largely on their having some taste which can be so utilised, such, for instance, as music. The possibility of errors of refraction may be remembered, as in the cases quoted on p. 968.

§ 539. Mental defect in children under 10 is a very different matter to the mental disorders of adults. Moral perversion or insanity, as already mentioned (p. 961), is not infrequent, but mania, melancholia and other adult varieties are extremely rare. Mental deficiency as a whole is the characteristic of this age period, and this is what will concern us now. It may be CONGENITAL, *i.e.*, due to ante-natal causes, or ACQUIRED (non-congenital), *i.e.*, due to causes arising after birth.

The *symptoms* and causes of these two groups differ considerably. In both varieties the children are "backward," deficient in all the faculties of the mind in greater or less degree; they carry evidences of this in their manner and behaviour, and sometimes but not always in their face. CONGENITAL cases always present some well-marked *alteration in the facial or cranial and bodily conformation*, and very often they are dwarfed in body as well as in mind. Children belonging to the NON-CONGENITAL class of mental deficiency do *not present these physical alterations*; their expression is bright, their cranium and face natural, their limbs well made, and excepting in the paralytic class they can generally walk and run well. Unfortunately they are also distinguished from the congenital class by a mobility and restlessness which is an indication of the difficulty of fixing their attention, and therefore of teaching them. They may also be distinguished from the congenital cases by a history of their having been mentally sound at birth and for some years afterwards, but among the lower orders a history of any diagnostic value is often wanting.

The practical point to ascertain in all cases of mental deficiency in childhood is their teachability, and this depends on two questions:—(1) Can their attention be attracted and fixed, and (2) are they imitative? (3) Their facial and bodily conformation should be observed; (4) the history or evidences of paralysis noted; and (5) if they have been to a Board school in England, the standard they have reached—the average in the infants' school is anything under 5 years; in standard I., 7 to 8 years; with an increment of one year for each succeeding standard, standard VII. being reached by normal children at 13 or 14.

The **congenital varieties of mental deficiency** in childhood (idiocy) date from birth, and the children generally present evidences in the facial and cranial conformation as well as in the mind. Some change in the shape of the hard palate is common to a good many of these cases.

The palate is too high, or V-shaped, too narrow from side to side, or otherwise deformed. The palate described by Dr. Clouston, and called by him the "neurotic palate," is one midway between the normal palate and the V-shaped palate, and is found in persons of nervous temperament, who are liable to hysteria, neuralgia, and migraine. Dr. Fletcher Beach found that out of 700 feeble-minded children 28 per cent. had V-shaped or otherwise deformed palates, and 60 per cent. had neurotic palates. On the other hand a high or V-shaped palate does not necessarily imply congenital deficiency, as was at first thought.

The VARIETIES OF IDIOCY (congenital mental defect in childhood) as given by Dr. Fletcher Beach,[1] are six in number.

1. *Simple congenital idiocy* includes children *without any obvious* abnormality of the cranium, or limbs, only in the face or palate (*supra*). In some the facial expression may be fairly intelligent, but most of the lower grade present an animal expression, thick lips, pug-nose, large coarse ears, broad, thick, depressed bridge of nose, narrow or hairy forehead, and underhung jaw. The *Mongol* or *Chinese* type of congenital deficiency is so called from the resemblance of the face to that of the Chinese, the palpebral fissures sloping downwards and outwards. They may be regarded as "unfinished" children, as they are often born of mothers who have suffered from continued ill-health during pregnancy; sometimes they are the youngest of a large family, or born of parents advanced in life. These children are very imitative and therefore educable. Practically, all of this class make good progress under proper education.

2. *Microcephalic* idiocy includes children whose heads are smaller in circumference than normal, which averages about 19 inches. The head may measure 17, 15, or even 12 inches; the forehead is narrow, and slopes backwards, corresponding with the deficiency of the frontal development of the brain. The features are frequently normal, eyes large, and nose aquiline. These children rarely make much improvement, for they have but little power of attention, though some of them are imitative. The question of craniectomy was raised some years ago in connection with these cases, as the small size of the brain was thought to be due to premature union of the cranial sutures, but this is now known to be incorrect. It is due to an arrest of growth of the whole nervous system.

3. *Scapho-cephalic idiocy* is so called from the boat-shaped character of the head, the antero-posterior being considerably longer than the transverse diameter. This is rare; Beach speaks of having seen only three or four cases. Other distortions of shape in the head met with are the asymmetrical head (flattened on one side), the sugar-loaf head, etc. But too much dependence must not be placed on the shape of the head unless it is combined with other factors. The children are generally deficient in intellect, but teachable.

4. *Hydrocephalic* idiocy occurs in some of the children born with hydrocephalus. Some retain their intelligence for a considerable while, but usually they drift into imbecility.

5. In *paralytic* cases the child has had hemiplegia, paraplegia or diplegia *dating from* birth. The paralysis may be due to some inflammatory condition of the membranes in utero, or pressure or injury before or at the time of birth (birth palsy, *q.v.*). Cerebral atrophy, congenital malformations and other lesions have been found. Mentally, these children generally make good progress under education, but physically little can be done for them.

6. In *Sporadic* and endemic CRETINISM, the markedly stunted growth is the most characteristic feature. The head is usually large, flat at the top, spread out at the sides (Fig. 6, p. 36). Hair coarse and dry, like that of a horse's tail. Voice, hoarse and squeaky. Speech, generally limited to a few words, and often monosyllabic. Their temperament is placid and good-natured. Under treatment by thyroid gland or extract, these cases make very remarkable progress, as in the two illustrations given. The treatment must be continued during the patient's lifetime; otherwise they relapse into their old condition.

Causes of CONGENITAL MENTAL DEFICIENCY in children. *α.* Causes acting *in the parents before birth* (heredity) occupy a very prominent place. Beach and Shuttleworth have exhaustively investigated this question in 2,400 cases. Abnormal conditions in the mother during pregnancy account for as much as 29 per cent., among which may be mentioned injuries, worry, anxiety, fright, or illness. Phthisis in the parents accounts for 28 per cent,, insanity and imbecility 21 per cent., epilepsy and other neuroses 20 per cent., and intemperance in the parents 16 per cent. Consanguinity acted as a cause in only 4 per cent., even when consanguinity in the grand-parents was taken into account. This is somewhat at variance with the generally supposed evil of marriage between blood relations; but it is a fact about which there can be no doubt, and, Dr. Beach adds, "Even in those cases where consanguinity was present, there were other hereditary tendencies, of themselves quite sufficient to produce the affection (congenital mental deficiency) without the presence of consanguinity." Syphilis in the parents was responsible for only 1·7, the effect of syphilis in the parents apparently being not so much the procreation of idiots as of children apparently healthy at birth whose nervous systems break down at the age of puberty.

β. Among the causes acting *at the time of birth*, prolonged or tedious parturition is undoubtedly the most potent. It is sometimes supposed that injury by forceps is a potent cause of the production of congenital mental deficiency, but it is worthy of note that in only 3 per cent. of Beach and Shuttleworth's cases was there a history of delivery by forceps.

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[1] *Clinical Journal*, Aug. 4, 1897; and "The Treatment and Education of Mentally Feeble Children," Fletcher Beach, 1895, Churchill, London.

His facts go to show that it is far better to put on the forceps early than to allow labour to be prolonged indefinitely. Such prolongation leads to great compression of the cranium, the brain being crushed, distorted, and otherwise injured. Such children when born are very often in an absolutely helpless condition. Others are subject to convulsions, and when death ensues, meningeal haemorrhage and cortical laceration are very often found.

Acquired (or **non-congenital) mental deficiency in children** under 10 is rarely difficult to differentiate from the congenital deficiency (*vide* p. 966). The varieties depend upon the causes. Among the *causes*, infantile convulsions was in operation in 27 per cent. of the 2,400 cases which Dr. Beach collected, epilepsy and other cerebral affections in 8 per cent., head injury in 6 per cent., fright or shock in 3 per cent., febrile diseases such as scarlatina, measles, whooping cough, enteric, small-pox, in 6 per cent., and over-pressure at school was only mentioned in 0·16 per cent.

1. *Infantile convulsions* if only occurring once or twice, may have no evil result, but if continued for some time throughout the early months of life, they generally result in a certain degree of weakmindedness. Many such cases occur in the children of insane or epileptic parents. They generally make little or no improvement, and drift into imbecility.

2. *Epilepsy*, as a rule, does not commence until after 10 years of age, but in some rare cases where it starts quite early in life it is attended with marked mental deficiency, although the fits may be few in number. Some cases improve if the fits cease, but mostly they go from bad to worse. Infantile convulsions cease before 2 years, epilepsy never occurs till after 2, and very rarely till after 14 years.

3. In *paralytic cases*, the mental deficiency is associated with hemiplegia and very often epileptiform seizures (*q.v.*) which have started *after birth*. They are mostly due to cerebral hæmorrhage and the scar which results.

4. *Traumatic causes.* Injuries to the head in childhood frequently result in mental deficiency. The injury as previously mentioned may have occurred during childbirth (compare β. above), though the symptoms may not have arisen until later. But quite as often the traumatism occurs after birth, and its history is a little difficult to make out. I frequently see mentally defective children in the out-patient room in whom careful inquiry reveals the history of a fall from a perambulator or cot which had been disregarded by the mother. One should always suspect an injury (perhaps concealed by the nurse) when the mental symptoms are associated with spastic hemiplegia. But very often there is no sign of paralysis, or scar on the head, and the fits or mental defect from which the patient suffers did not supervene till many months or a year or so after the injury, being in fact due to the cerebral scar which results.

5. *Inflammatory cases.* The acute specific fevers, meningitis, and other inflammatory conditions, may be followed by mental deficiency. It seems to me probable that most of these cases are due to concurrent or consequent inflammation of the brain or its membranes. The mental symptoms do not necessarily follow directly after the illness which has caused the mental defect as in the case about to be mentioned, and therefore we may have only the history to rely upon. These cases are among the worst which we have to deal with. They are analogous to cases of mania occurring in the adult, excepting in their universally unfavourable course. In October, 1897, a little girl, æt. 3½, was brought to me by her mother. She ran perpetually around the room from object to object, resting her attention on nothing for more than a second or two at a time; taking no notice of anybody. The mother said she was excitable at night and often mischievous. The birth had been natural, and the child normal at birth and up to the age of 2 years. Then she was laid up with a feverish attack, which was called "influenza," attended by very severe headache, with which she was ill for several weeks. She recovered completely, and it was not until the age of 3 that she began to be unmanageable and destructive, and the present condition slowly supervened.

6. *Hypertrophy of the brain* is included by Dr. Beach under the heading of inflammatory cases, because the post-mortem appearances of patients who died of this disease under his care showed that there was or had been chronic inflammation of the brain. In these cases both the brain and the skull are enlarged, the shape of the head being more of a square than in hydrocephalus.

7. *Hereditary syphilis.* Among the symptoms of this protean disorder is mental deficiency in childhood. It does not seem to affect the brain in infancy or early childhood, but about the period of the second dentition, or between that and the time of puberty, the nervous system sometimes shows signs of progressive degeneration. Sometimes this is manifested by disseminated sclerosis or other spinal disease, sometimes only by a steady deterioration of the mind.

8. *Reflex causes.* Mental symptoms of a pronounced kind arise in association with asthenopia in children. Two little girls aged 6 and 13 years were brought to me for great despondency, and crying without cause. The mother of each stated the child was

constantly crying without a reason, and was constantly saying that her parents and others were against her and hard on her. No heredity was in operation. The vision was found by my colleague, Mr. Work Dodd to be $\frac{6}{12}$ and $\frac{5}{12}$ of normal and on this being adequately corrected by glasses the mental symptoms entirely disappeared.

9. Theoretically, it is possible for some of the forms of mental alienation met with in adult life to appear in childhood, but mental disease practically only arises in childhood when one of the above causes is in operation.

The *prognosis* of ACQUIRED MENTAL DEFICIENCY in childhood is always very grave. Indeed, there are few cases where much improvement takes place when once the deficiency is established. If the mental symptoms seem to have come to a standstill, and the child is in any degree educable, something may be done in this direction; but as a rule less can be done for acquired cases than for congenital, and their tendency is to get worse.

In the TREATMENT OF MENTAL DEFICIENCY IN CHILDHOOD, whether CONGENITAL or ACQUIRED, a certain amount can be done by education and discipline. The first and fundamental principle is to remove the child from its home and from the surrounding conditions which have been unconsciously adapted to its defective habits. The second point is to fix the child's attention, and this can best be done by employing music, pictures, and other things which please and attract the child. It may be taken as a general principle that the educability of a child, that is to say the prospect of recovery, depends entirely upon the facility with which the attention can be attracted, and the efficiency with which it can be retained when once secured. The shape of the head and aspect of the face cannot be relied upon; the brightest looking children are often the most volatile. On the other hand, the dull-looking children who take an interest in their work will make more progress. As a rule the prognosis is much worse in irritable restless cases than in quiet cases. It is usual to commence with the education of the senses—touch first, then sight, hearing, taste, and smell. Speech also has to be educated by means of speech drill. Any vicious habits must be gently corrected, and punctuality and discipline enforced. Later on, perseverance, and ideas of justice, duty, self-reliance, prudence, and forethought have to be inculcated. The progress of any case under treatment depends very much on the age at which it is commenced. It should always be begun as early as possible, and the idea which some mothers have of the symptoms passing off at 7 or 14 by an abrupt change from mental enfeeblement to mental brightness should be met and combated as wholly fallacious, and very damaging to the prospects of success.

GROUP III. INTRACRANIAL INFLAMMATION.

§ 540. Pyrexial disorders of the nervous system are not numerous, and are practically confined—if acute anterior poliomyelitis and certain exceptional cases of cerebral hæmorrhage be omitted—to acute inflammation of the meninges. They consist of the following intracranial inflammatory conditions, all of which present *cerebral symptoms*, *pyrexia*, and its attendant symptoms. The onset in most is acute, though in some it is insidious.

I. Tuberculous meningitis.
II. Acute meningitis.
III. Post-basic meningitis.
IV. Epidemic cerebro-spinal meningitis (p. 623).
V. Intracranial abscess.
VI. Sinus thrombosis (especially septic thrombosis).
VII. Hæmorrhage, chronic degenerative diseases, etc.

CLINICAL INVESTIGATION. 1. The *cerebral symptoms* in intracranial inflammation may be grouped into those of irritation and compression. The symptoms of cortical *irritation* which are the most usual, especially in the early stages, consist of headache, vomiting, tonic or clonic spasms or convulsions, sleeplessness, restlessness, delirium, quick pulse, and contracted pupils. The symptoms of *compression*, which usually ensue later, are mental dulness, paralysis of the limbs and cranial nerves, a slow full pulse, dilated or unequal pupils, and stupor passing on to coma. The earlier or irritative stages may have to be diagnosed from other causes of convulsions (§ 591): the later compression stage from the typhoid state (§ 347) or coma (§ 525).

2. *The scalp and cranial bones* should be examined for any swelling or tenderness on percussion—*e.g.*, any œdema (sinus thrombosis), erysipelas or Pott's puffy tumour (an œdematous swelling of the scalp which is usually a sign of subcranial, extra dural, abscess in that situation). When there has been a *wound of the scalp*, the skin may have healed up externally though pus has formed beneath, and the infective products carried along the perivascular sheaths or lymphatics into the skull. But the patient may go about for days or weeks before symptoms of intracranial inflammation present themselves.

3. The *ear* and *sinuses* near it, the nose, and the pharynx should also be carefully examined for any discharge or signs of disease.

4. The *cranial nerves* need to be examined severally (§ 602 *et seq.*). Inflammation like tuberculous meningitis, which has a predilection for the base nearly always reveals cranial paralysis at some time; but those which affect the convexity of the brain, such as simple meningitis, give rise rather to convulsions.

5. The *lungs* and other organs should be examined; many cases of tuberculous meningitis are secondary to tuberculosis of the lungs or peritoneum.

6. The *age* and *history* of the patient, especially as regards any previous otorrhœa, may be noted. Acute meningitis may occur at any age, and runs a course of about 2 days to 2 weeks: tuberculous meningitis chiefly affects children, and runs a course of 2 weeks to 2 months (including premonitory stage): post-basic meningitis is confined to infants under 1 year and runs a prolonged and indefinite course.

7. The *temperature* should be carefully investigated, and a chart obtained—

diurnal intermissions suggest tuberculous meningitis (§ 541);
a nondescript or continued temperature suggests acute meningitis (§ 542);
the temperature falling after a few days of initial rise and becoming subnormal suggests cerebral abscess (§ 543);
wide and irregular intermissions suggest septic sinus thrombosis (§ 544);

§ 541. I. Tuberculous meningitis, tuberculosis of the cerebral meninges, is the commonest cause of intracranial inflammation in children. The cerebral pia mater, especially at the base and in the Sylvian fissure, becomes studded with grey miliary tubercles. It is more frequent in children, and in the male sex. Tuberculous meningitis may (i.) supervene in a case of tuberculosis elsewhere of which previous symptoms have existed; (ii.) it may be ushered in primarily and suddenly with vomiting and convulsions in a child previously in good health, or (iii.) it may come on insidiously as a primary affection of the meninges, with a long prodromal period.

The *symptoms* are usually insidious and variable. There is a prodromal and 3 other stages, the latter running a course of 2 or 3 weeks and passing imperceptibly into each other. In the *prodromal* stage, which may last a considerable time, the child gets thinner, loses its appetite, is peevish and listless, but with intervals of brightness, when the mother thinks it is becoming well again. Intermittent pyrexia may be revealed from time to time. (1) The *irritative* stage is ushered in very often suddenly, with (i.) severe headache, vomiting, or convulsions, and there may be delirium, and the child may lie curled up, shrinking from light or sound. (ii.) There is a moderate degree of fever, usually of a diurnally intermittent type. (iii.) The pulse is quick, and the pupils contracted. (iv.) The head generally shows a characteristic retraction with rigidity of the muscles of the neck. (v.) The abdomen is retracted. When vomiting is present in a child, without diarrhœa, tuberculous meningitis or other head mischief should be suspected. (vi.) The peculiar cry, known as the "hydrocephalic cry," may accompany this stage of the disease, and last till the end. As a rule the irritative stage lasts only a few days to a week. (2) The *compression* stage is announced by symptoms pointing to paralysis of the cranial nerves, as in all diseases situated chiefly at the base of the skull. (i.) The pupils are unequal, and become dilated, and strabismus is common owing to paralysis of the 6th or 3rd nerves. (ii.) The patient is drowsy, with slow pulse, which may be irregular, and sighing respiration. (iii.) There is vaso-motor paralysis, indicated by the red streak when the finger nail is drawn along the skin. This condition is known as the "tâche cérébrale" (Trousseau) from the idea, now known to be mistaken, that it was found only with cerebral lesions. (iv.) Optic neuritis or tubercle of the choroid may develop. (3) The *final* stage is marked by increasing irregularity and weakness of the pulse and the respiration. The drowsiness passes into coma, and there is incontinence of urine and faeces. Mucus gathers in the bronchial tubes and breathing is laboured, and often takes the Cheyne-Stokes form. Just at the end the temperature may rapidly fall, or it may rise very high (106° or 107°), and local paralyses, *e.g.*, of the arm or leg, or ptosis may occur, with convulsions. This stage lasts only a few days.

Tuberculous meningitis in adults is often secondary to tubercle in the lungs, abdomen or elsewhere. It differs from the disease in children in

(1) the advent is most insidious and prolonged. A case is referred to in the footnote to p. 611, where headache and intermitting pyrexia were the only symptoms for nine weeks, until within two weeks of death, when optic neuritis and signs of tuberculous mischief at the apex of the lung appeared. (2) Severe and persistent headache with intermitting pyrexia are always very prominent features. Hemiplegia or rather hemiparesis may be the earliest and principal symptom, and aphasia is not infrequent because of the invasion of the Sylvian fissure by the tubercle, while (3) optic neuritis, strabismus or other evidence of cranial nerve paralysis, such as facial paresis, or inability to swallow, are present as in children. Vomiting and convulsions are rare. (4) The course lasts from 5 to 12 or more weeks.

Diagnosis. *Enteric fever* with head symptoms, pain and delirium, and no rash or diarrhœa, may for a week or longer be mistaken for meningitis; and on the other hand the meningitis which has little headache may be mistaken for enteric fever. But the course of the disease and the aid of the Widal reaction soon reveal its true nature. Any one of the *specific fevers* may be ushered in with headache so severe as to give rise to a suspicion of meningitis; but in all such cases the headache ceases when delirium begins. Irregularity in the breathing and the pulse, and evidences of basal paralyses are conclusive signs of meningitis. *Simple meningitis* is distinguished from the tuberculous variety by its rapid course, which rarely exceeds a few days, by there being no basal paralyses, and by the presence perhaps of a local cause, such as injury or otitis media. A rapidly growing *tumour* (*e.g.*, tuberculous or gliomatous) may simulate meningitis. Here the character of the optic neuritis present aids diagnosis—if very intense, with swelling and hæmorrhages, it is probably due to tumour. The early stages of tuberculous meningitis in young women may resemble *hysteria*, but the eyes should be examined for papillitis, and it is well to remember that divergent strabismus even if transient, or unequal pupils, are evidences of organic disease. *Post-basic meningitis* occurs in infants under 1 year and has a longer course. In *marasmic conditions* in children drowsiness and convulsions may arouse the suspicion of tuberculous disease being present, but in such cases the fontanelle is depressed because the intracranial pressure is not raised, local paralyses and fever are absent, and the subsequent course reveals the condition.

Prognosis and treatment. Tuberculous meningitis is essentially a chronic disorder, though its average duration varies considerably

—from three weeks to three months are about the limits. Until recent years it was regarded as fatal, though cases of undoubted recovery have been published (Trousseau, Rilliet, Carrington). Curiously enough the treatment which has been most successful consists of calomel and pot. iod., remedies which are directed against syphilitic lesions, and it seems probable that some of the alleged cases of recovery may have been syphilitic meningitis. The prognosis depends mainly on three things, first, the period of the disease at which the patient comes under treatment; secondly, the height and range of the temperature which is the measure of the activity of all tuberculous processes; and thirdly, the extent of the tuberculous mischief in other organs. Symptomatic *treatment* is employed as in other head diseases; thus ice-bags are applied to the head to relieve the pain. Bromides or chloral internally, or evaporating lotions to the shaven scalp also relieve the headache. Mercurial or iodoform ointment rubbed in to the scalp is recommended.

§ 542. II. **Acute meningitis** (leptomeningitis: arachnitis: acute hydrocephalus) is a diffuse inflammation of the pia mater and arachnoid (leptomeningitis), simple or purulent according to its exciting cause, chiefly affecting the convexity of the brain.

Symptoms. There are symptoms and signs of cortical *irritation* (§ 540), followed by signs of *compression*, *i.e.*, increased intracranial pressure (*vide* § 540). The inflammation affects the convexity rather than the base of the brain, the reverse of tuberculous meningitis, and the symptoms consist, therefore, of muscular twitchings, spasms, sometimes convulsions, and later on paralysis of the muscles of the body, rather than paralysis of the cranial nerves as in tuberculous meningitis. There is no prodromal stage, the invasion being sudden. The course of the disease lasts usually a few days to 1 or 2 weeks. (1) The temperature remains continuously high, and when pyogenic causes are in operation there may be rigors and wide variations. Other symptoms are—(2) severe persistent headache; (3) delirium; (4) retraction of the head, rigidity of the muscles of the neck, diffuse hyperæsthesia and pain, especially about the neck, from involvement of the spinal nerve roots. Optic neuritis and ocular paresis are rare. (5) There may also be vomiting, flushing of the face and conjunctivæ, the "tâche cérébrale" of Trousseau, and herpes on the lips or face. Kernig's sign (§ 514) is present if the inflammation extends to the spine. (6) There is usually a history or evidence of some cause, for the disease is rarely primary, the most common cause being disease of the petrous bone, with or without ear disease, attended by a purulent discharge.

Varieties and *causes.* Acute meningitis may, as just mentioned, be purulent or non-purulent. The former is accompanied by greater constitutional disturbance, perhaps by rigors; and the course is more rapid and fatal than in the non-purulent variety.

(a) *Simple* or *non-purulent meningitis* may be (i.) *secondary* to constitutional disorders such as the specific fevers, pneumonia, influenza, diphtheria, acute rheumatism, measles, small-pox, erysipelas, scarlet fever, anthrax, gonorrhœa, and actinomycosis, also, it is said, to gout, chronic nephritis and heart disease in their terminal stages. (ii.) A *primary* meningitis may be due to a pneumococcus found in the cerebro-spinal fluid, without pneumonia being present. A primary cerebro-spinal meningitis also occurs in the sporadic and epidemic form, due to the diplococcus intracellularis (§ 623). *Tuberculous* and *post-basic* meningitis are special forms described respectively in § 541 and below.

(b) *Purulent* or *pyogenic meningitis* may occasionally arise in association with the above conditions. But the most frequent cause of purulent meningitis is (1) extension of inflammation from adjacent parts either from *without*, *e.g.*, in caries or necrosis of the cranial bones, erysipelas of the face or scalp; or from *within*, *e.g.*, a cerebral abscess or sinus thrombosis. Any of the bones may be involved, but disease of the petrous bone is the most frequent cause, with or without otitis media: nasal or ethmoid disease is a much less common cause. In disease of the cranial bones, a chronic localised thickening of the meninges may prevent a localised collection of pus, a subcranial abscess, from spreading laterally; but the edges are liable at any time to become softened, and so permit of a generalised purulent meningitis. (2) Punctured wounds of the scalp may cause meningitis in the same way as in cerebral abscess formation (*q.v.*). (3) Pyæmia and malignant endocarditis may be attended by a septic purulent, and rapidly fatal form of the disease. (4) The pneumococcus is probably one of the commonest causes of suppurative meningitis.

Diagnosis. Acute meningitis has to be diagnosed from other forms of meningitis and from *acute specific fevers*, pneumonia and other acute diseases, which may simulate meningitis at their onset with headache, vomiting, and absence of local signs. *Pneumonia* may be very deceptive at first before lung signs are definite, but the rapidity of respiration affords us a clue. *Tuberculous* meningitis has a more insidious onset, a prodromal stage and a prolonged course. When there is otitis media, meningitis may require to be differentiated from *cerebral abscess*, which has more localised symptoms, a low temperature after the first day or so, and the headache lasts longer before the onset of coma. In septic *sinus thrombosis* the temperature is marked with recurring rigors, and local signs of the sinus involved are usually seen, such as the brawny swelling in the neck in lateral sinus thrombosis. The diagnosis of the *cause* also requires investigation, especially in children. When due to otitis the patient usually holds the hand to the head. Lumbar puncture (§ 622) and bacterial cultures may aid us in the diagnosis of the cause.

Prognosis. In diffuse pyogenic forms of meningitis the disease usually runs a fatal course in a few days, but a simple meningitis is more favourable—it may completely recover, or if traumatic in origin, it may pass into a chronic localised meningitis. Recovery has been reported after the primary pneumococcus form, though this and the other septic forms are generally fatal.

Treatment. The patient must be kept in a dark room, perfectly quiet, the head shaved, and an ice-bag applied. A purge should be given, preferably of calomel. Some recommend blisters, or the cautery to the back of the neck. Only milk diet is allowed. Mercury, especially in the form of inunction, is recommended together with large doses of iodide of potassium. The cause must be carefully investigated, and where local causes are in operation, such as injury or disease of the scalp. these must be treated surgically. In children and infants especially, puncture of the tympanum with consequent outlet of a little pus has often resulted in prompt relief of symptoms of intracranial irritation. Lumbar puncture (§ 622) not only affords a clue to diagnosis of the primary malady, but may by the relief of tension result in recovery.

III. **Posterior basic or chronic infantile meningitis** is a disease occurring in infants under 12 months, due to inflammation in the posterior fossa of the skull associated with a special diplococcus, the diplococcus of Weichselbaum. This is the same microbe as that of epidemic cerebro-spinal meningitis (§ 623), and post-basic meningitis is now (1905) generally regarded as a sporadic, sub-acute form of that disease. The exudation glues together the cerebellum and medulla, leading ultimately to the blocking of the foramen of Majendie and distension of the ventricles (hydrocephalus). The characteristic *symptoms* are—(1) the gradual onset of the retraction of the head, which may amount to opisthotonos of the spine with flexor and extensor spasm of the limbs ; (2) vomiting ; (3) rigidity of the limbs, which may be general or localised to one extremity ; (4) paroxysms of high fever lasting a day or two. The disease occurs in infants from 3 to 12 months old, only occasionally in older children. It is diagnosed from tuberculous meningitis by the age of the patient, the usually greater degree and persistence of cervical opisthotonos, the longer course of the disease, and the absence of optic neuritis in post-basic meningitis.

Prognosis. The disease often runs a very prolonged course of weeks or months. Death has taken place after 19 months. Cases may undoubtedly recover ; Drs. Lees and Barlow say 1 in 6 recover completely. In others grave sequelæ remain : hydrocephalus resulting from the distension of the ventricles ; blindness (without optic neuritis) ; deafness (with consequent loss of speech) ; and defective intelligence ; or a combination of these.

Treatment. Mercurial ointment should be rubbed into the neck, and iodide of potassium in doses of 1 to 3 grains every 2 hours is believed to cause absorption of the exudation. Surgical treatment has been carried out, but with little success in the resulting hydrocephalus.

IV. **Epidemic cerebro-spinal meningitis** has already been described on p. 632.

§ 543. V. **Intracranial abscess.** Abscess of the brain may occur in an acute or chronic form.

The symptoms of cold or *chronic abscess* are identical with those of any other cerebral tumour apart from the history and the symptoms referable to the condition which caused it. In quite a number of cases the history of a cause is wanting, and such cases present only the features of intracranial tumour (*q.v.*). The commonest cause of cerebral abscess undoubtedly is suppurative middle ear or bone disease.

The *symptoms* of acute abscess of the brain may be divided into three stages. The *initial* or inflammatory stage lasts from 12 hours to three days ; in some cases it has lasted a week. Very often this stage may not be noticed ; and in most instances the patient does not come under observation until it is over. The three chief symptoms which characterise this stage are—*pain*, *vomiting*, and *rigors*. (i.) The pain in the head may be burning, shooting, and continuous or intermittent, and there is tenderness over the seat of the abscess. (ii.) The vomiting occurs without nausea, and has no relation to food. (iii.) The rigors vary greatly in severity, from a slight shivering to a shuddering. If the rigors are frequent there is probably present some systemic infection or sinus thrombosis (*q.v.*). The temperature in this stage is above normal, but not high. If the disease be due to otitis media, the discharge stops. The *second or collapse* stage[1] shows symptoms similar to the first stage, only much less marked. (i.) The pain is diminished; the patient may lie quietly moaning with the hand over the affected part. Tenderness is brought out on percussion, and is useful in localising the site of the mischief. (ii.) Vomiting and vertigo occur on movement only. (iii.) Cerebration is slow; the patient will answer questions correctly, but only after long delay, and he may fall asleep in the middle of a sentence. (iv.) The temperature in this stage is normal or subnormal, if the abscess is uncomplicated. (v.) The pulse is slow and full (30 to 60). Respiration is slowed. (vi.) Optic neuritis develops; and (vii.) paralyses may occur which aid in diagnosing the site of the lesion. This stage lasts from one to five weeks. In the *third* or paralytic stage the abscess terminates in one of three ways: (i.) by compression symptoms—deepening stupor, coma and death. (ii.) The abscess may open on the surface of the brain, leading to meningeal symptoms with high temperature, quick pulse, vomiting, convulsions, etc. (iii.) The abscess may open into the ventricles of the brain, which is characterised by lividity, dilatation of the pupils, stertorous breathing, high temperature, convulsions, coma, and death within 12 hours.

Diagnosis. Abscess of the brain has to be diagnosed from *acute meningitis*, in which the symptoms are more of an irritative character, with high temperature and quickened pulse. In *sinus thrombosis*, there are recurring rigors and local signs of the involvement of the sinus. It must be remembered that both meningitis and thrombosis may accompany abscess, and in this case the symptoms are confusing. If meningitis and abscess occur together the presence of the latter will be indicated by a slower pulse than would be met with in uncomplicated meningitis. *Intracranial tumours* resemble abscess. The slow progress of the symptoms, the greater amount of optic neuritis and more definite focal phenomena, with the absence of rigors, and of any source of abscess formation, are in favour of a diagnosis of tumour; while the presence of a subnormal temperature is in favour of abscess.

Causes of cerebral abscess. Many valuable observations have been made in this domain within the last few years by Sir William Macewen. Briefly, the causes of cerebral abscess may be said to belong to three categories—disease or injury of the cranium or scalp; pyæmic infection from the scalp or other parts; and softening of a tuberculous mass (very rare). The locality of the abscess depends a good deal upon its source. The chief causes are:—1. *Middle Ear Disease* is by far the most common cause. The abscess occurs in the temporo-sphenoidal lobe, or less commonly in the cerebellum. There is usually necrosis or tuberculous disease (caries), or occasionally syphilitic disease of the petrous bone. (i.) The inflammatory process spreads through the meninges, over the tegmen tympani (roof of the tympanum), and if the local adhesions be present to prevent lateral extension, the process goes on, even without any erosion of the bone, to ulceration of the brain, and consequent pus formation within the brain substance. (ii.) Or cerebral abscess may form in the white matter, the grey matter of the cortex of the brain remaining uninvolved. Here the inflammation spreads along the perivascular sheaths of the vessels or along the veins entering the brain. Briefly, the foregoing means that abscess may occur, (a) by contiguity, or (b) by extension along the lymphatic sheath of the vessels, or along the veins from a distance. 2. *Disease of the antrum of Highmore or nasal bones* causes abscess in the frontal lobe. 3. *Injury such as compound fracture*, with necrosis of the bone, may cause an abscess, the position of which depends upon the seat of the injury. 4. *Wounds of the scalp or abscess*, where the skin is healed but there is pus formation beneath the bone. 5. *Carbuncle of the face* (rare), or *scalp* causes abscess when septic matter passes by the facial vein and pterygoid plexus, or by the ophthalmic vein, to the cavernous sinus. 6. *Disease of the orbit* causes abscess by a similar method of extension. 7. *Erysipelas* (rare) more often causes leptomeningitis. 8. *Abscess of pyæmic origin* may occur in any part, but the most common situation is the occipital lobe. The pyæmic sources are septic embolism (*e.g.*, in malignant endocarditis), and pyæmia.

Prognosis and *treatment.* Early diagnosis and correct localisation of this serious condition is most important, for surgical treatment is successful but untreated cases usually die. In some cases the abscess undergoes apparent cure, and, if small, it may remain encapsuled for years, or may even dry up. Macewen mentions a few cases where the abscess discharged externally through eroded bone. The usual course of an acute uncomplicated abscess is from two to six weeks. A chronic abscess may last months or years, but is always a source of danger; after remaining without symptoms for years, it may suddenly burst into the meninges or into the ventricles. Uncomplicated abscess if discovered in good time is readily amenable to *treatment* in the hands of a skilled surgeon; but if symptoms of meningitis or sinus thrombosis accompany it, the prospect is much more grave, especially if the meningitis be diffuse.

§ 544. VI. Sinus thrombosis. thrombosis of the cerebral sinuses, may be PYOGENIC and accompanied by pyrexia and stupor, or NON-PYOGENIC, accompanied by stupor only.

a. In **pyogenic sinus thrombosis** there is pyrexia of a pyæmic type, *i.e.*, wide variations with rigors. Any one of the sinuses may be involved, but the lateral sinus (secondary to suppurative middle ear or temporal bone disease) is the favourite position.

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[1] Some call this the *latent* stage, but Macewen objects to the use of such a term in reference to a condition where symptoms are present.

The *symptoms* common to septic thrombosis of any of the sinuses are (i.) Severe headache, vomiting, and high fever of a pyogenic type accompanied by rigors and sweats (see chart, p. 645); (ii.) optic neuritis supervening in a day or two, and perhaps photophobia; (iii.) drowsiness deepening into coma, and if prompt operative measures be not adopted, death ensues.

The *localising signs* of the particular sinus thrombosed depend on the extension of the inflammation along the corresponding extracranial veins, and on the presence of the cause. Thus in *lateral sinus* thrombosis there are pain and tenderness in the mastoid region, together with the other signs of a suppurative otitis media or disease of the petrous bone; the inflammation spreads down the course of the jugular vein on the same side, and backwards behind the mastoid; and consequently there is generally some hard brawny swelling in these positions; if there has previously been a discharge from the ear it mostly ceases. When the *longitudinal sinus* is thrombosed the localising signs consist of œdema of the scalp, distension of the veins over the forehead, and sometimes strabismus, associated with convulsions at the onset. This is the sinus which is most often affected by non-pyogenic thrombosis, and when pyogenic in origin the cause is usually some septic lesion of the face or scalp. When the *cavernous sinus* is affected the localising signs are œdema of the eyelids and root of the nose, sometimes also of the pharynx, exophthalmos, and paralysis of the 2nd, 3rd, 4th, ophthalmic division of the 5th, and 6th, nerves. Pyogenic thrombosis of this sinus may arise from some septic lesion of the orbit, nose, pharynx, or face.

The *diagnosis* of pyogenic sinus thrombosis from *septicæmia* is very difficult unless the local signs are pronounced. *Acute meningitis* is also differentiated with difficulty, but here we get a lower temperature without rigors and sweats, and retraction of the head and neck. In *cerebral abscess*, the temperature is normal or subnormal after the initial rise, and it is accompanied not so much by stupor as by headache and paralysis of the cranial nerves.

The *prognosis* of pyogenic sinus thrombosis is exceedingly grave, unless it is promptly dealt with by surgical measures, such as trephining over the mastoid cells, tying the jugular vein below, cutting down upon the lateral sinus, and turning out the clot. With these measures, cases may recover, not without.

b. **Non-pyogenic sinus thrombosis** gives rise to coma (§ 525) mainly in marasmic conditions. It is met with among infants suffering from prolonged diarrhœa; among the aged; and less frequently in adults in the last stages of exhausting diseases, such as phthisis or cancer. The superior longitudinal is the sinus most often affected. Headache, passing on to drowsiness and coma, are the leading symptoms; the temperature may be slightly raised, but it never forms a prominent feature. In adults delirium may mark the onset, but the condition is difficult to diagnose with certainty. In infants the coma supervenes almost imperceptibly upon the drowsiness which accompanies their exhausted condition. In children, epistaxis and convulsions should make one suspect the condition even in the absence of localising signs. The localising signs are mentioned under septic sinus thrombosis.

The *prognosis* is grave, especially in adults. Children may recover, but only with impairment of intellect.

Treatment. The patient should be kept at rest with the head and shoulders slightly raised. The neck must not be bent, so that the blood be not hindered in its return. Tonics and stimulants may be given.

VII. Pyrexia, usually of short duration, may accompany certain **intra-cranial lesions of acute onset,** such as (i.) hæmorrhage, especially into the pons, when there is sudden coma, contracted pupils and hyperpyrexia (see apoplexy, § 525a); (ii.) lesions occurring in the course of chronic degenerative disease of the central nervous system, such as disseminated sclerosis and G. P. I.

GROUP IV. MOTOR DISORDERS OF THE NERVOUS SYSTEM.

Motor disorders of the nervous system may consist of PARALYSIS (*below*), INCO-ORDINATION (§ 571), INCREASED MUSCULAR ACTION (§ 576), or MUSCULAR ATROPHY (§ 594).

a. PARALYSIS.

Paralysis may take the form of—

Hemiplegia	§ 545
Paraplegia	§ 549
Brachiplegia	§ 562
Monoplegia and single nerve paralysis	§ 563
Generalised paralysis	§ 568

The patient complains of weakness or paralysis *of the limbs on* ONE SIDE OF THE BODY—the case is one of HEMIPLEGIA, which may be either of sudden onset or of gradual onset.

§ 545. **Hemiplegia,** paralysis of one side of the body, is due to a lesion of the internal capsule or some other part of the motor tract of one hemisphere, somewhere above the decussation in the medulla.

The CLINICAL INVESTIGATION of a case of hemiplegia comprises an examination of the paralysed limbs, the cranial nerves, the cardiovascular system, the urine and the viscera, and a study of the history and mode of onset. Our chief object is to ascertain the *nature* and *position* of the lesion.

Symptoms. In 9 cases of hemiplegia out of 10 the loss of power in the limbs is on the same side as that in the face; when it is on opposite sides it is known as "crossed hemiplegia," and the lesion is situated in the pons. The arm is usually more affected than the leg, and the leg than the face. Only the lower part of the face is affected, the food collects round the teeth on that side, but the patient can close both eyes. If the tongue is paralysed, during protrusion it is turned to the affected side, due to the unbalanced action of the healthy genio-hyoid and genio-hyoglossus. Hemianæsthesia on the paralysed side is not present unless the hinder limb of the internal capsule is also involved. There may be an initial or *early* rigidity, coming on at the outset and lasting a few hours (as in cases of hæmorrhage), or coming in a few days' time and lasting for a few weeks (as in cases of irritative lesion). There is also a *late* rigidity, which comes on in all cases of hemiplegia after a few weeks or months and lasts permanently, and is due to descending lateral sclerosis.[1] In the course of years the muscles may undergo a certain amount of *organic contracture*, due to intrinsic changes in the muscular substance, and the hemiplegic muscles are also liable to various kinds of tremor, mobile spasm, and athetosis. These are especially common after the hemiplegia of childhood. The other features are those of an upper neuron paralysis, viz., (1) the deep reflexes are increased; (2) there are no electrical changes; and (3) there is no wasting beyond that of disuse. Finally, there are *vaso-motor*

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[1] Rare cases have been recorded in which the paralysis remained flaccid (hémiplegie flasque of Bouchard).

alterations in hemiplegia. There are known to be centres in the cortex, irritability of which produces coldness and paleness of the limbs. If the lesion producing the hemiplegia causes irritation of these centres, the hemiplegic limbs are paler, colder, and bluer. If, on the other hand, it gives rise to suppression of function, then the paralysed limbs are congested, sweating, and sometimes œdematous. These considerations explain the apparently contradictory observations made as to general dropsy, which sometimes avoids and sometimes is present in excess in the paralysed limbs, and the observation that the pulse is sometimes smaller, sometimes larger in the paralysed arm, and, finally, of the fact that the pupil is generally contracted on the paralysed side, either from paralysis of the sympathetic or from irritation of the third cranial nerve. In all organic diseases of the brain which produce hemiplegia, there is generally more or less mental disturbance, and this is particularly marked in cortical lesions, such as tumours of the cortex, hæmorrhages on to the cortex, and various meningeal affections, particularly when the front of the brain is involved. Aphasia (§ 528) is associated with right hemiplegia in many cases owing to the involvement of Broca's convolution ; in left-handed persons left hemiplegia may be so associated.

The chief *causes of hemiplegia* (10 in number) may be conveniently divided into those of *sudden onset*, *i.e.*, in the course of a few minutes or a few hours, and those of more *gradual onset*, *i.e.*, in the course of days, weeks or months.

A *sudden onset* always indicates a vascular lesion—

- Injury.
- Hæmorrhage.
- Embolism.
- Thrombosis.

In hysterical hemiplegia also the onset is generally sudden.

A *gradual onset* may be due to—

- Intracranial tumour.
- Abscess.
- Chronic cerebral meningitis and pachymeningitis.
- Altered blood states (chorea, diphtheria, anæmia, pregnancy, etc.).
- Chronic degenerations of the nervous system.

The *age* of the patient is an important aid in the diagnosis of the nature of the lesion in hemiplegia.

In the first half of life suspect in order—Embolism; Thrombosis from syphilitic endarteritis; Hysteria (in females); Tumour; Abscess; Hæmorrhage in childhood (occasionally); Post-febrile and other blood conditions.

In the second half of life in order of frequency we get—Hæmorrhage; Thrombosis from arterial degeneration; Tumour; Aneurysm; Chronic degenerations of the nervous system.

Syphilis plays a very important part in the causation of hemiplegia, which may be produced in several ways: (1) a syphilitic endarteritis and thrombosis, (2) the formation of a gummatous tumour, (3) meningeal affections, or (4) aneurysm.

a. The **sudden causes** (vascular lesions) are much more frequent than those of gradual onset.

(1) INJURY TO THE HEAD may produce hemiplegia by depressed bone, extravasated blood, or, later on, by the formation of an abscess or a cicatrix. It is known by (i.) the history of injury; (ii.) by being preceded or accompanied by loss of consciousness, stupor, or convulsions; and (iii.) early rigidity in the affected limbs.

(2) HÆMORRHAGE (apoplexy, § 525a) from rupture of a vessel or an aneurysmal dilatation occurs most frequently in the lenticulo-striate artery (Fig. 159, p. 981) in the internal capsule, though it may occur anywhere. Its differential characters are as follows:—(i.) The onset is usually very sudden, with coma, and often convulsions, the degree of these symptoms depending on the locality and the extent of the hæmorrhage; occasionally, when the hæmorrhage is small, headache, giddiness, or vomiting alone mark the advent. (ii.) The patient is usually of middle or advanced life, and often presents a history of chronic interstitial nephritis, though cerebral hæmorrhage is also known to occur in children. (iii.) The rigidity of the affected limbs comes on with the hemiplegia, and is succeeded by late rigidity and sometimes by tremor. Meningeal hæmorrhage rarely occurs unless the meninges are previously unhealthy, as in general paralysis.

(3) EMBOLISM (§ 525a), blocking of a cerebral artery, often of the middle cerebral artery, may arise from a fragment of a vegetation from a cardiac valve, or by some septic or other embolus. It causes a localised cerebral ischæmia and softening.

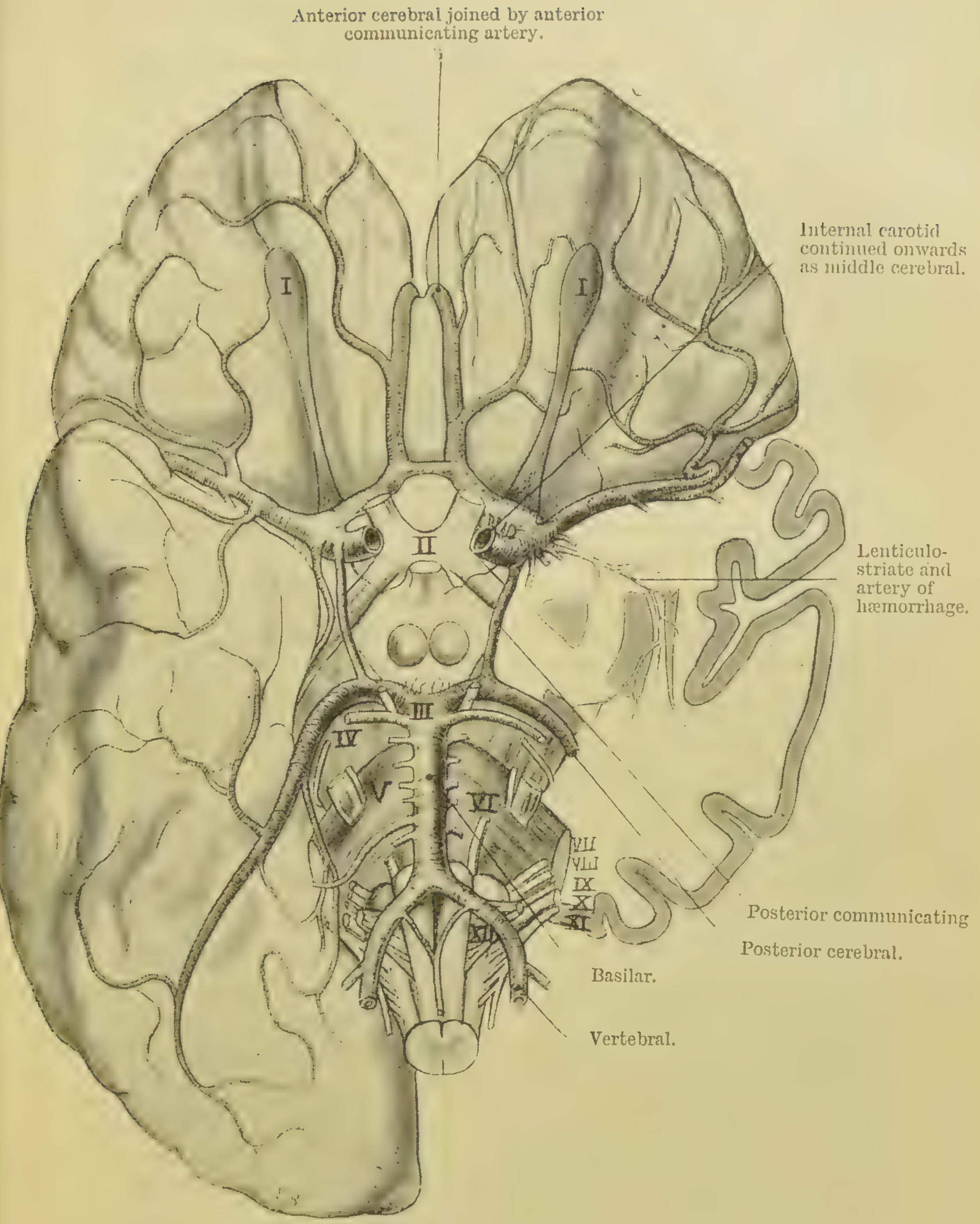
Anterior cerebral joined by anterior communicating artery.
Internal carotid continued onwards as middle cerebral.
Lenticulo-striate and artery of hæmorrhage.
Posterior communicating
Posterior cerebral.
Basilar.
Vertebral.
I
I
II
III
IV
V
VI
VII
VIII
IX
X
XI
XII

Fig. 159.—The BASE OF THE BRAIN, showing the arterial distribution and the cranial nerves. In the oblique section of the left hemisphere are seen from without inwards—grey matter of the island of Reil; claustrum (grey); external capsule (white); lenticular nucleus (grey); internal capsule (white) with artery of hæmorrhage; and caudate nucleus (grey). I. Olfactory nerve; II. optic chiasma; III. bifurcation of vertebral artery between the third nerves; IV. (on right crus cerebri) beside fourth nerve; V. (on pons varolii) beside fifth nerve; VI. sixth nerve (abducens); VII. facial nerve; VIII. auditory nerve; IX. glosso-pharyngeal nerve; X. vagus or pneumogastric; XI. spinal accessory; XII. hypoglossal nerve.

It is differentiated by—(i.) sudden hemiplegia, accompanied and often preceded by severe headache and giddiness; but (ii.) rarely by convulsions, and as a rule consciousness is not lost; (iii.) the patient is often young; (iv.) there is often a history of rheumatic fever, and cardiac valvular disease, especially mitral stenosis; (v.) the muscles are generally flaccid at first, and the hemiplegia is more often on the right side, because of the continuity of the left middle cerebral artery with the aorta.

(4) ARTERIAL THROMBOSIS (§ 525a) consists of coagulation of the blood within an artery owing to its occlusion by disease of the wall or a thrombotic condition of the blood as in phthisis. Senile change in the wall of the artery, associated with cardiac enfeeblement, is its commonest cause. In young subjects syphilitic endarteritis is very often in operation. Venous thrombosis is not necessarily associated with hemiplegia; it is referred to under coma. Thrombosis of an intracranial artery is recognised by—(i.) sudden advent of hemiplegia, without unconsciousness as a rule; (ii.) the paralysed muscles are flaccid at first, and there is no early rigidity; (iii.) the patient is mostly advanced in years, unless the disease be syphilitic.

Arterial occlusion is the commonest of the cerebral SYPHILITIC LESIONS and in that way becomes the commonest cause of hemiplegia under 40. A syphilitic endarteritis gradually occludes an artery, and results in thrombosis and "softening" of a circumscribed area, just as does atheroma of the cerebral vessels. The symptoms of a syphilitic softening resemble atheromatous thrombosis except in two particulars, namely, (1) the extent of the softening and therefore of the paralysis is often more limited (*e.g.*, one arm or one leg); and (2) the patient is generally young or middle-aged; a thrombotic hemiplegia in a person under 40 or 45 is almost certainly syphilitic. This kind of cerebral syphilis is distinguished from the meningeal lesions or gumma by the absence of irritative signs; the advent, contrary to what one would expect from the *gradual* occlusion of an artery, is always more or less sudden, unaccompanied by unconsciousness, convulsions, delirium, rigidity of limbs, optic neuritis or paralysis of cranial nerves.

(5) HYSTERICAL HEMIPLEGIA is recognised by the following characters—(i.) the advent is usually sudden, and often dates from an emotional shock or hysterical seizure; (ii.) the paralysis is usually flaccid, incomplete in degree, and the face is exempt; (iii.) it is often accompanied by hemianæsthesia of the same side, sensitive and sensorial; (iv.) the condition varies from day to day, and may disappear suddenly, unexpectedly and completely; (v.) the youth and sex of the patient are characteristic, and she presents other evidences of the hysterical diathesis (*q.v.*).

b. The **gradual onset** of hemiplegia, in the course of several weeks or several months, points to one of the following conditions—intracranial tumour, abscess, chronic cerebral meningitis (and pachymeningitis), altered blood states, or chronic degenerations of the nervous system. Nevertheless tumour, aneurysm and abscess may sometimes run a latent or prolonged course and then suddenly develop hemiplegia.

§ 546. (6) **Intracranial tumour** may exist for a long time without any symptoms, especially if the tumour be situated in the frontal region, but it is generally characterised sooner or later by a progressive and insidious hemiplegia. As regards the nature of the tumour, syphilitic gumma is one of the most frequent in adult life, and is specially apt to affect the meninges at the base or over the convolutions. It is well to bear in mind that malignant tumours may occur primarily in the brain; cancer is confined to advancing years, but sarcoma occurs quite early in life. A tuberculous nodule is only met with in the young, and a history or the existence of tubercle elsewhere point to the tumour being of this nature. An aneurysmal tumour is not common. The *symptoms* of intracranial tumour vary very considerably with its locality (which will be discussed § 547), but most cases present the following *symptoms in common*—(i.) Constant headache, vertigo, and causeless vomiting from time to time, precede and accompany the illness, especially if the tumour be situated near the cortex. (ii.) Jacksonian epilepsy (§ 591) is also frequent if the tumour be situated in or near the cortex. (iii.) Œdema of the optic discs, leading to atrophy, is present in a considerable proportion of the cases (five-sixths, Sir Wm. Gowers[1]). For the occurrence of these the tumour need not be near the optic nerve; it is believed to be due to increased intracranial pressure. The two symptoms, headache, and a marked degree of optic neuritis, are alone very strongly suggestive of tumour of the brain. (iv.) Paralysis of other cranial nerves, depending on the position, but especially of the 6th, 4th, and 3rd, owing to their relatively long course within the cranium. (v.) Incomplete hemiplegia coming on slowly and insidiously but progressively, with rigidity and the other features of the upper neuron paralysis; accompanied by other symptoms according to the situation of the tumour (see § 547). Mental apathy and delayed cerebration are usually present.

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[1] "The Diagnosis of the Nature of Organic Brain Disease," Phonographic Medical Library, vol. i., 1897.

(7) ABSCESS OF THE BRAIN is rarely primary, and may arise (1) from direct extension, as from disease of the cranial bones, syphilitic, tuberculous or after injury, or following disease of the frontal or nasal sinuses. (2) Abscess also arises from septic blood conditions—pyæmia, emboli from malignant endocarditis, or from the lungs when affected with tubercle, bronchiectasis, gangrene, or abscess. It has followed influenza and the specific fevers. The symptoms of cerebral abscess resemble those of tumour, and differ according to the site. Abscess of the temporal lobe after middle ear disease is not uncommon, and in this case the hemiplegia is of slow onset, is ill-defined, and the face is involved. The other symptoms are described in intracranial inflammation, § 513. Constitutional symptoms are only found at the onset and the conclusion of the case.

(8) CHRONIC MENINGITIS and PACHYMENINGITIS (fibrous thickening of the meninges) sometimes occur (though rarely) as a solitary lesion. The condition is mostly due either to chronic alcoholism, syphilis, or injury, and some say that in the absence of these, chronic meningitis may be excluded. Tuberculous meningitis may also cause hemiplegia, especially in children. (i.) The advent is usually gradual: (ii.) the degree of the hemiplegia is slight: (iii.) optic neuritis is frequently present: (iv.) very generally some of the cranial nerves are involved as they pass through the membranes—lesions of the sensory nerves causing pain, disease of the motor nerves causing atrophic paralysis with R. D. (v.) There are always some signs of mental alteration, and not infrequently epileptiform convulsions from irritation of the cortex. Indeed, epilepsy occurring for the first time in a person of 30 or 40, is very probably due to syphilitic disease of the meninges.

Syphilitic meningeal affections may take the form of a chronic meningeal thickening or a gummatous deposit, either primarily, or secondarily to osseous lesions. These are distinguished clinically by severe and continuous headache, and the presence of irritative signs.

(9) ALTERED BLOOD STATES, such as those due to chorea, severe anæmia, pregnancy, typhoid fever, influenza, scarlet and other fevers, may give rise to hemiplegia. It is possibly due to thrombosis, or to more transient vascular changes. It is recognised by—(i.) the antecedent history, and (ii.) the incompleteness and transitory nature of the paralysis.

(10) Hemiplegia may also form a minor feature in the course of several CHRONIC DEGENERATIVE CONDITIONS of the nervous system, such as disseminated sclerosis, general paralysis of the insane, and bulbar paralysis, the diagnosis of which is dealt with elsewhere.

Prognosis of hemiplegia. If the paralysis has been considerable, one can hardly hope for complete recovery. The most favourable kinds, both for recovery and for life, are those due to injury, hysteria, and an altered blood state. Next in order comes syphilis, which, if diagnosed early and treated thoroughly, may generally be cured. All the other causes are more serious. Localised cerebral tumours are sometimes removable, and life may be prolonged, but the paralysis rarely disappears entirely. Abscess is capable of surgical treatment. The most unfavourable kinds of hemiplegia are those due to embolism, thrombosis, and haemorrhage, the last-named being the gravest of all; and the usual history in these cases, even if the patient recover, is that a recurrence takes place during the ensuing year or two, in which the patient dies. As regards symptoms, it is usual for the leg to recover before the arm; if the converse of this happens, the prognosis is certainly less favourable, as showing that the damage is too great for the substitution of function which leads to the recovery of the leg taking place. The formation of blisters or sloughs over the gluteus is of bad prognostic significance; the general condition of the patient, and the existence of some other disease, such as chronic granular kidney, may also constitute unfavourable circumstances.

Treatment of hemiplegia. In hæmorrhage, the patient should at first be kept at perfect rest; the treatment of hæmorrhage, embolism, and thrombosis should be directed to the cardio-vascular system (see p. 942). Intracranial tumour and abscess, unless we can positively exclude syphilis, should be put through a full course of iodide and antisyphilitic remedies while the locality is being accurately diagnosed with a view to surgical interference should these remedies fail. For aneurysm, full doses of iodide of potassium are also useful, and other remedies directed to control the circulation. Chronic meningitis, if of a syphilitic nature, should be treated accordingly; if due to injury, and the position of the focus of the disease can be diagnosed, surgery may be invoked. As regards the treatment of the paralysed limbs, it is usual to regard them as hopeless, but a great deal can be done by the judicious application of massage, galvanism, and galvano-faradism. The first improves the nutrition of the muscles considerably, the second and third maintain and improve their functional activity. Galvanism only may be applied to rigid muscles, and general hygienic measures will aid. Electricity should not be started till two months after a cerebral hæmorrhage, and should be stopped if headache, faintness or fatigue appear. If the motor tract is definitely destroyed nothing will restore its functions completely, but a good deal may be done by the means just mentioned and by the education of other centres to take on the functions of those destroyed. Strychnine must be avoided when the muscles are rigid.

§ **547. The localisation of intracranial lesions** may be considered, first, in regard to certain recognised types of paralysis; secondly, certain groups of symptoms corresponding to the three basal fossæ; and thirdly, the symptoms pointing to positions in the encephalon.

A. Lesions in certain positions give rise to certain TYPES OF PARALYSIS.

(1) Hemiplegia is usually due to a lesion in the internal capsule.

(2) "Crossed paralysis" (face on one side, body on opposite side) is typical of a lesion in the lower part of the pons.

(3) Paralysis of the 3rd nerve on one side, with hemiplegia or hemiataxy on the opposite side, is typical of a lesion in the crus cerebri.

(4) Conjugate deviation of the eyes indicates a lesion at or above the ocular nuclei.

B. Intracranial tumours in the—

POSTERIOR FOSSA—resemble symptoms of pontine and medullary tumours, but without conjugate deviation. Cranial nerves from VI. to XII. may be involved (Fig. 159, p. 981).

MIDDLE FOSSA—lesion of one third nerve, and symptoms of involvement of crus cerebri.

ANTERIOR FOSSA—paralysis of nerves entering the orbit, loss of sight and smell on side of lesion.

C. Destructive lesions in different positions in the encephalon are indicated as follows—

FRONTAL LOBE—mental torpor and depression, sometimes exophthalmos on the side of a tumour, and perversion of smell. Usually no motor or sensory disturbance.

OCCIPITAL LOBE—altered field of vision (hemianopia); if bilateral, blindness; if involving left angular gyrus, word blindness.

TEMPORO-SPHENOIDAL LOBE—deafness; if in left superior convolution, word deafness; if tip, taste and smell affected.

CORPUS CALLOSUM—(i.) gradual onset of hemiplegia, with vague hemiplegic symptoms on other side; (ii.) mental torpor; (iii.) coma and death without involvement of cranial nerves; (iv.) headache and vomiting rare throughout (Bristowe).

CORTEX (see Figs. 150, 151, p. 885)—*Rolandic area*, Jacksonian convulsions, starting in part controlled by affected area. Paralysis of opposite side, flaccid at first; monoplegia, rarely complete hemiplegia; impaired muscle sense; ataxy in finer movements (*e.g.*, fastening buttons). If *left inferior frontal* involved, aphasia (motor speech). The convulsions are preceded by aura of flashes of light, if occipital cortex; by noises or music, if superior temporo-sphenoidal cortex; by odour, if tip of temporo-sphenoidal cortex.

INTERNAL CAPSULE—hemiplegia, face and body, of opposite side. When posterior part involved, hemianæsthesia, hemianopia, disturbed hearing, tremor (especially when optic thalamus also involved), hemichorea and athetosis. In a linear (antero-posterior) lesion, paralysis of face and leg, arm escaping.

OPTIC THALAMUS—hemianopia; hemianæsthesia; post-hemiplegic chorea. Cannot with certainty be distinguished from lesions involving the sensory part of internal capsule and optic radiations. Paralysis of emotional movements of face together with post-hemiplegic choreic disorders are in favour of a lesion involving opposite thalamus.

CORPORA QUADRIGEMINA—*Superior corpora*: nystagmus, loss of pupillary contraction to light and accommodation, ataxy if fibres to medial lemniscus involved. *Inferior corpora*: auditory disturbance, partial deafness of both and especially of opposite ear. Defective mastication from involvement of motor root of V., and the 4th nerve is sometimes involved. Lesions of corpora quadrigemina simulate locomotor ataxy or cerebellar tumour. Ophthalmoplegia and reeling gait, especially if associated with bilateral defective hearing, render diagnosis probable.

CRUS CEREBRI:—simultaneous onset of III. nerve paralysis on side of lesion, and of hemiplegia on opposite side, is very characteristic; and if *tegmen* involved, hemiataxy of opposite side.

PONS:—very varied symptoms; most characteristic are combination of paralysis of V., VI. and VII. on side of lesion, and of body on opposite side. *Upper part of pons*: paralysis and anæsthesia of face and body on side opposite to lesion. *Lower part of pons*: "Crossed paralysis," face on side of lesion, body on opposite side; VI. and VII. paralysed together; conjugate deviation to side away from destructive lesion; in acute lesions, contracted pupils and hyperpyrexia.

MEDULLA:—Difficulty in articulation and swallowing, associated with disturbances of heart and respiration, and paresis or paralysis of limbs on one or both sides. All cranial nerves from VIII. to XII. affected. Often secondary to cerebellar or pontine disease.

CEREBELLUM:—Ataxy (reeling), static and dynamic, not affected by closing eyes; movements fairly co-ordinated when in bed. Vertigo. Nystagmus. Paresis of trunk or limbs when pressure on pyramidal tracts. K. J.'s sometimes absent. Hydrocephalus by pressure on veins of Galen leading to distended ventricles.

§ 548. **Hemiplegia in children** is met with in congenital and in acquired forms, the latter being usually under 10 years old. The child has a sudden fit of convulsions, followed by coma and hemiplegia. In such cases athetosis and post-hemiplegic chorea are common sequelæ, and Jacksonian epilepsy is also seen. The affected limbs do not grow so well as the others, though the muscles are not really wasted, and some may be hypertrophied from the constant movement in athetosis. The mental condition in such patients varies—it may be normal or merely dull; others, especially the congenital cases, are idiots, with squint.

The *prognosis* as to recovery is bad, but the duration of life is not affected by this disease except in the congenital and mental cases, who frequently die in early life from intercurrent diseases.

Etiology. Congenital hemiplegia occurs from (i.) injury at birth, or protracted labour causing meningeal hæmorrhage; (ii.) porencephalus (cavities in the brain); and (iii.) atrophy of one hemisphere. Acquired hemiplegia comes on in the majority of cases before the 2nd year, rarely after the 10th year. The hemiplegia in such cases is apt to come on during or after an acute specific fever, and has been found after death to be due to embolism, thrombosis, or hæmorrhage. Tuberculous meningitis and tumour, cerebral abscess, polio-encephalitis superior, and trauma are also causes of hemiplegia in children.

Treatment. The possibility that infantile convulsions are associated with cerebral lesion should be remembered and bromides and a purge should be given. The hemiplegia may profitably be treated by electricity and massage: the rigidity and contractures are treated by massage and exercises.

The patient complains of weakness or **paralysis** *of* BOTH LEGS—the disease (general debility being excluded) is PARAPLEGIA, which may be of an upper neuron or rigid type; a lower neuron or flaccid type; or, a functional or variable type.

§ 549. **Paraplegia** is paralysis of both lower extremities. It is most often due to some structural or functional disease of the spinal cord, for it is here that the motor tracts of the two sides of the body run side by side and can therefore be affected by a single lesion. Paraplegia may also be due to disease of the peripheral nerves.

CLINICAL INVESTIGATION. Assuming that the case is really one of paraplegia and not simply a stiffness of the joints due to gout, rheumatism or old age, it is desirable (1) to investigate the history of the case, (2) to examine the spinal column, and (3) to ascertain whether the paralysis conforms to the upper neuron type, the lower neuron type, or the functional or variable type.

(1) The history of the case throws considerable light on the nature of the lesion, and particularly in regard to the mode of onset and evolution; the history of pain; and the age of the patient. The onset is very rapid

in vascular lesions or those of an acute inflammatory type. The onset takes place more gradually in some forms of compression paraplegia, such as Pott's disease (§ 550), in multiple neuritis, chronic myelitis, and syringomyelia. The history of pain in the spine or legs is of importance, and some observers even go so far as to divide paraplegias into painful and painless. The presence of pain indicates an involvement of the nerve trunks or the posterior nerve roots, as in meningeal affections and tumours. Paraplegia in *childhood* in a large proportion of cases is due to Pott's disease or infantile palsy. *Adults* from 20 to 40 are chiefly affected by paraplegia due to multiple neuritis, hysterical or syphilitic paraplegia. In *advanced* life the slow degenerative lesions are more often met with.

(2) Local examination of the spine should never be omitted. It will teach us at a glance whether or not angular curvature or malformations be present: percussion down the spine may elicit tenderness as in Pott's disease, tumour or meningeal affections. Vertical pressure upon the head while the patient is sitting or standing will cause severe pain if vertebral caries or cancer be present, but not in hysterical or neurasthenic conditions. Rigidity or restricted movement accompanies all painful organic affections of the spine, as in meningeal diseases.

(3) It will be found convenient for clinical purposes to make three groups of paraplegias—

(*a*) In **upper neuron**, spastic or **rigid paraplegias**, the paralysis is persistent and progressive, the muscles are rigid, with no tendency to atrophy beyond that due to disuse, the deep reflexes are increased, and there are no electrical changes . § 550 *et seq.*

(*b*) Paraplegias of the **lower neuron** type are **flaccid**. In this type the paralysis is persistent and continuous from day to day, the muscles are atrophied, the deep reflexes are absent or diminished, and there is the reaction of degeneration . § 556

(*c*) In the **functional** or **variable paraplegias** the paralysis is less pronounced and less persistent than in the foregoing: it is apt to vary from day to day, and there is neither atrophy nor electrical changes . . . . § 558

(*a*) The **upper neuron, spastic,** or **rigid paraplegias** form the largest of the three groups, and it contains a most important sub-group, viz., the compression paraplegias. The three features of this kind of paraplegia are those which belong to all paralysis due to lesions of the upper neuron, viz., rigidity, increased deep reflexes, no marked muscular wasting, and no alteration of the electric reactions. The members of this group differ from the functional paraplegias in their steady and usually progressive course. The following are the causes of the spastic paraplegias placed more or less in order of frequency—

Compression Paraplegias.

I. Compression paraplegia due to Pott's disease.
II. Compression paraplegia due to extra-medullary tumours.
III. Injury.

Inflammation of the Cord or its Membranes.

IV. Myelitis and its varieties.
V. Hæmorrhage (spinal and meningeal).
VI. Embolism.
VII. Chronic spinal pachymeningitis.

Chronic System Lesions (Sclerosis).

VIII. Disseminated sclerosis, General Paralysis of the Insane and tabes dorsalis—occasionally.
IX. Primary lateral sclerosis.
X. Ataxic paraplegia and Friedreich's disease (occasionally).
XI. Amyotrophic lateral sclerosis.
XII. Infantile cerebral and spinal paraplegia and other double cerebral or cerebellar lesions.
XIII. Toxic sclerosis.

Malformations.

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§ 550. **Compression paraplegia** is perhaps the commonest form of paraplegia, and is due to the compression of the spinal cord either by spinal caries (Pott's disease), or other disease or injury of the vertebra, or a tumour pressing upon the spinal cord.

The *symptoms* common to all forms of compression paraplegia, in addition to those just named belonging to upper neuron paraplegias, are (1) pain at some time from pressure upon the nerve roots, and generally speaking it may be said that when the pain precedes the paraplegia the lesion is extradural, when it is *vice versâ* the lesion is probably intradural. (2) The symptoms are apt to be more marked in one leg than in the other (with the notable exception of Pott's disease).

I. **Vertebral caries** (Pott's disease, tuberculosis of the spine) may be regarded as the type of compression paraplegias. It is one of the commonest causes of paraplegia in young persons. It is the commonest of the compression paraplegias, injury coming next, and vertebral cancer next. The paraplegia in Pott's disease is due to the pressure of the inflammatory products which accumulate outside the dura mater and compress the cord. Its differential features are :—(1) In the incipient stage the child probably complains of "stomach ache" or pains in the region of the umbilicus, the back or the loins, due to pressure on the nerve roots. (2) The onset of the paralysis is usually gradual and is rarely quite complete, but on rare occasions the onset is rapid. Both legs are affected equally. (3) There is marked rigidity and increase of the deep reflexes, often twitchings of the muscles. (4) In the early phases jarring by percussion on the head or jumping on the heels causes pain; there is generally pain and tenderness of the spine, and in course of time other indications appear at the seat of the mischief. (5) The subjects of the disease are mostly children or young persons who have a family or antecedent history of tuberculosis and perhaps other manifestations of that disease. (6) The course is always protracted. Spinal caries even without operation tends in the long run to become quiescent, and if the patient is able to undergo prolonged rest it is wonderful how the use of the legs may be restored even after complete paralysis. I have seen cases recover after a complete paralysis lasting 2 or 3 years, and have made autopsies on patients who could walk whose cord at one spot was no larger than a quill. The *diagnosis* is only difficult when there is no curvature or other local indication of caries. Of the other causes of extradural compression paraplegia cancer and aneurysm are the commonest. They occur in older subjects and are associated with great increase of the pain on movement. Exostoses can only be suspected when found also in other parts of the body. These and all the other causes of compression paraplegia are distinguished from Pott's paraplegia by (1) the unilateral predominance of the symptoms; and (2) pain being relatively a more prominent symptom. For the diagnosis of the position of the lesion see below.

II. **Compression paraplegia due to spinal tumour.** Tumours of the spinal cord may be (α) extra-medullary (arising outside the spinal cord), or (β) intra-medullary (arising within the spinal cord). Extra-medullary tumours are the more common and it is these which produce compression paraplegia.

α. EXTRA-MEDULLARY SPINAL TUMOURS may be extra- or intra-thecal, but these two cannot be clinically distinguished. The following are the principal extra-medullary tumours—syphilitic gumma, sarcoma, carcinoma, tuberculous growths, myxoma, fibroma, meningeal hæmorrhage, hydatid cysts, chondroma, exostoses, lipoma (rare), or neurofibroma (springing from the nerve roots in the canal). Aneurism, carcinoma, and sarcoma may arise outside the vertebral column and invade the cord after eroding the vertebræ, but these three lesions generally have their own proper symptoms.

Symptoms of extra-medullary spinal tumours. (1) Pain radiating in the distribution of the nerve trunks pressed on is usually the earliest indication and is rarely absent; generally it is one-sided, round the chest or abdomen, or down the leg; sometimes there is pain at the site of the tumour. (2) Progressive paraplegia starting usually in one leg and spreading upwards from the toes, more marked on the same side as the pain, associated with (3) anæsthesia having the same features. Finally both the paralysis and the anæsthesia become complete up to a definite level. The upper limit of the anæsthesia is the best guide as to the seat of the tumour, bearing in mind that the posterior roots enter the cord two or more inches (in the dorsal region) above their entrance into the spinal canal (Fig. 160), and therefore the tumour must be cut down upon well above the anæsthetic limit. Of the two sub-groups of extra-medullary tumours, extra-thecal lesions are more often attended by pain, and intra-thecal lesions by cramps and contractures of the muscles at night, indicating compression myelitis. The superficial abdominal reflexes are sometimes abolished on the same side as the tumour—the supra-umbilical in tumours between the 8th and 9th, the infra-umbilical in tumours lying between the 11th and 12th dorsal vertebræ. Sometimes the umbilicus is drawn towards the healthy side. In meningeal hæmorrhage the symptoms of pain and paralysis are of sudden onset.

The *diagnosis* of such cases rests on the presence and character of the pain, the progressive evolution of the symptoms, and their predominance on one side. Cervical meningitis has pain on both sides. The *prognosis* depends mainly on the nature and position of the tumour, and whether secondary myelitis has occurred. The treatment (apart from anti-syphilitic remedies) is surgical.

β. SYRINGOMYELIA AND INTRA-MEDULLARY TUMOURS (§ 601) sometimes come under notice for paralysis of the legs, and this paraplegia may be very peculiar, namely, an *atrophic* paralysis *associated with rigidity and increased knee-jerks.* The paraplegia is sometimes of an ordinary spastic type but as a rule this only occurs late in the disease. The earlier symptoms of syringomyelia usually appear in the arms (sensory and trophic changes). But the symptoms of an intra-medullary tumour necessarily differ with the position of the tumour and the column chiefly involved.

III. **Injury to the spinal column** may cause displacement or damage to the vertebra (fracture-dislocation), or hæmorrhage, or may initiate acute myelitis or meningitis (*q.v.*). The symptoms following injury are those either of destruction (paralysis), or irritation (spasm), and the history of the injury generally enables us to make a diagnosis.

In cases of transverse myelitis after fracture-dislocation it was formerly taught that the reflexes below the lesion were exaggerated unless and until the disease had, by descending, destroyed some part of the reflex arcs. But Dr. Carlton Bastian has shown that in cases of transverse myelitis the reflexes below the lesion may be absent, even without such extension downwards.

§ 551. The exact **localisation of spinal lesions,** (a) as regards its LEVEL, is effected in three ways—(1) by the distribution of the motor weakness; (2) by the alteration of the reflexes; and (3) by the level of the upper limit of the pain and anæsthesia (see Head's areas below). (1) and (2) are given in the table below.

(b) The particular COLUMN which is affected can be ascertained by studying the table in § 500.

FUNCTIONS OF THE SPINAL SEGMENTS.

SEGMENT OF CORD, AND SPINOUS PROCESS.	MUSCLES SUPPLIED.	REFLEX.	SENSATION.
II and III C. Opposite 1st C.	Sterno-mastoid. Trapezius. Scaleni and neck. Diaphragm.	Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex. Neck.
IV C. Opposite 2nd C.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra- and infra-spinatus. Levator ang. scapulæ.	Pupil reflex = 4th to 7th cervical; *i.e.*, dilatation of the pupil produced by irritation of neck.	Neck. Shoulder. Outer arm.
V C. Opposite 3rd C.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator longus and brevis. Rhomboids and teres minor. Pectoralis (clavicular part). Serratus magnus.	Scapular reflex = 5th cervical to 1st thoracic; *i.e.*, irritation of the skin over the scapula produces contraction of the scapular muscles. Tapping tendon of supinator longus in wrist produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm, front and back.
VI C. Opposite 4th C.	Biceps. Brachialis anticus. Pectoralis (clavicular part). Serratus magnus. Triceps. Extensors of wrist and fingers. Pronators.	Triceps reflex = 5th to 6th cervical; *i.e.*, tapping elbow tendon produces extension of forearm. Posterior wrist reflex = 6th to 8th cervical; *i.e.*, tapping tendons causes extension of hand.	Outer side of forearm, front and back. Outer half of hand.
VII C. Opposite 5th C.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapularis. Pectoralis (costal part). Latissimus dorsi. Teres major.	Anterior wrist reflex = 7th to 8th cervical; *i.e.*, tapping anterior tendons causes flexion of wrist. Palmar = 7th cervical to 1st thoracic; *i.e.* stroking palm causes closure of fingers.	Inner side and back of arm and forearm. Radial half of the hand.
VIII C. Opposite 6th C.	Flexors of wrist and fingers. Intrinsic muscles of hand.		Forearm and hand, inner half.
I D. Opposite 7th C.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar eminences.		Forearm, inner half. Ulnar distribution to hand.

FUNCTIONS OF THE SPINAL SEGMENTS—*continued.*

SEGMENT OF CORD, AND SPINOUS PROCESS.	MUSCLES SUPPLIED.	REFLEX.	SENSATION.
II to XII D. Opposite 1st to 10th D.	Muscles of back and abdomen. Erectores spinæ.	Epigastric reflex = 4th to 7th thoracic; *i.e.*, irritation of mammary region causes retraction of epigastrium. Abdominal = 7th to 11th thoracic; *i.e.*, stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen in bands running around and downward, corresponding to spinal nerves. Upper gluteal region.
I L. Opposite 11th D.	Ilio-psoas. Sartorius. Muscles of abdomen.	Cremasteric = 1st to 3rd lumbar; *i.e.*, stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum.
II L. Opposite 11th D.	Ilio-psoas. Sartorius. Flexors of knee (Remak). Quadriceps femoris.	Tapping patellar tendon causes extension of leg.	Outer side of thigh.
III L. Opposite 12th D.	Quadriceps femoris. Inner rotators of thigh. Abductors of thigh.		Front and inner side of thigh.
IV L. Opposite 12th D.	Abductors of thigh. Adductors of thigh. Flexors of knee (Ferrier). Tibialis anticus.	Gluteal = 4th to 5th lumbar; *i.e.*, stroking buttock causes dimpling in fold of buttock.	Inner side of thigh and leg to ankle. Inner side of foot.
V L. Opposite 12th D.	Outward rotators of thigh. Flexors of knee (Ferrier). Flexors of ankle. Extensors of toes.		Back of thigh, back of leg, and outer part of foot.
I to II S. Opposite 1st L.	Flexors of ankle, and toes. Peronæi.	Plantar reflex.	Back of thigh. Leg and foot, outer side.
III to V S. Opposite 1st L.	Perineal muscles.	Ankle-clonus. Bladder and rectal centres.	Skin over sacrum. Anus. Perinæum. Genitals.

Head's areas of analgesia and hyperalgesia (Figs. 160 and 161) are of use for the localisation of a spinal injury or lesion. They have been elaborated by the successive researches of James Ross,[1] James Mackenzie,[2] William Thorburn,[3] and more particularly Henry Head.[4] These are areas of diminished or increased *sensibility to pain*, which correspond with certain spinal segments as marked in the illustration. The reader is asked to remember in what follows, that we are not now concerned with tactile sensation, but only with sensibility to pain (algesia). A destructive lesion of a spinal segment or nerve root is attended by analgesia of the corresponding area, an irritative lesion by hyperalgesia. The sensibility to pain of the skin below the spinal lesion is of course disturbed, but to localise a spinal lesion you should carefully test the sensibility from above downwards with the point of a pin, and you will find an *abrupt margin of normal sensibility to pain* at the upper border of the affected area. The important question really is to what level does the sensibility to pain remain healthy. Head's areas were discovered mainly by the investigation, (1) of cases of injury to the spinal nerve roots, (2) cases of visceral disease associated with soreness of the skin or reflex pain, and (3) cases of herpes.

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[1] *Brain*, January, 1888, part x., p. 233, and "Diseases of the Nerv. System."
[2] *Brain*, part xvi., 1893, pp. 321 and 515; and *Med. Chronicle*, August, 1892.
[3] *Brain*, part xvi., 1893, p. 355.
[4] *Brain*, parts xvi., 1893, p. 1; xvii., 1894, p. 339; xix., 1896, p. 153.

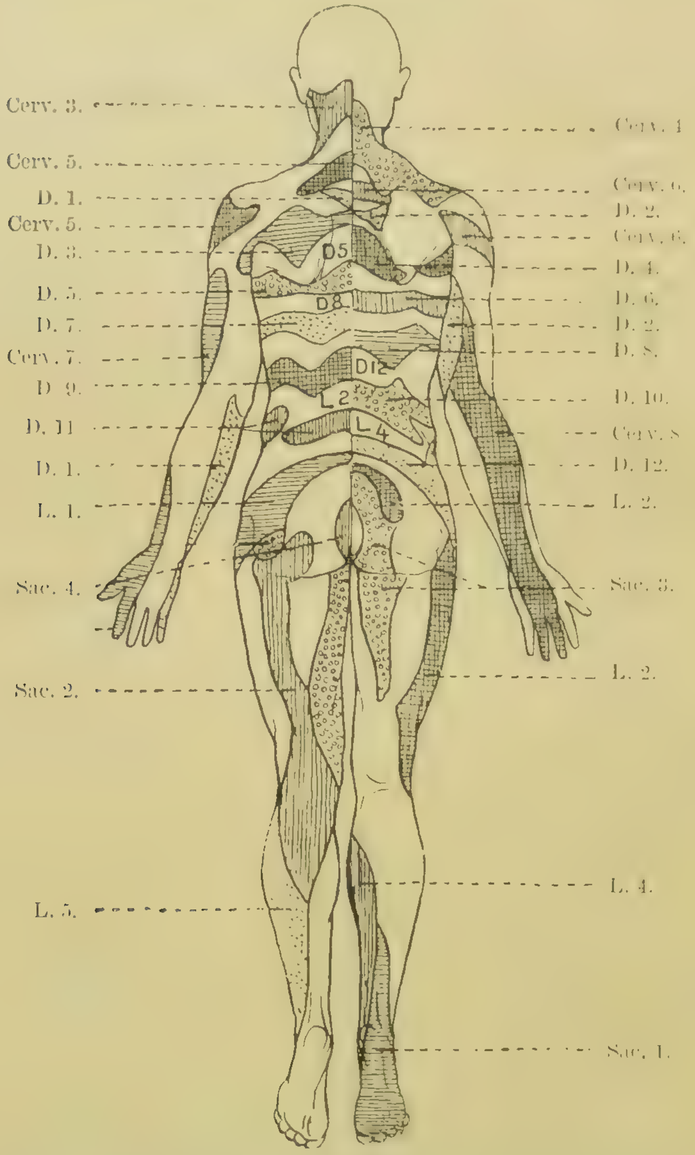

Fig. 160.—Segmental algesic areas of Head, *i.e.*, areas of increased or diminished cutaneous sensibility to pain when the corresponding spinal segment is irritated or destroyed respectively.

These areas may also have two other possible uses. In certain *visceral diseases* groups of these areas are apt to be the seat of tenderness, and even pain, which is probably of a reflex nature; single area tenderness is practically never met with. To test these areas the rounded head of a lady's hat-pin is best, its smooth surface evoking the sensation of soreness. The various organs stand in connection mainly with the areas as shown in table below, the areas being those indicated in the figures. (Dr. Henry Head in Quain's Dictionary of Medicine, 3rd Ed., p. 1138.) There are also certain smaller *spots* of maximum tenderness to which a patient chiefly refers his pain, which remain tender longer than the rest of the skin around. The whole of this question is at the present time one of more scientific than practical interest.

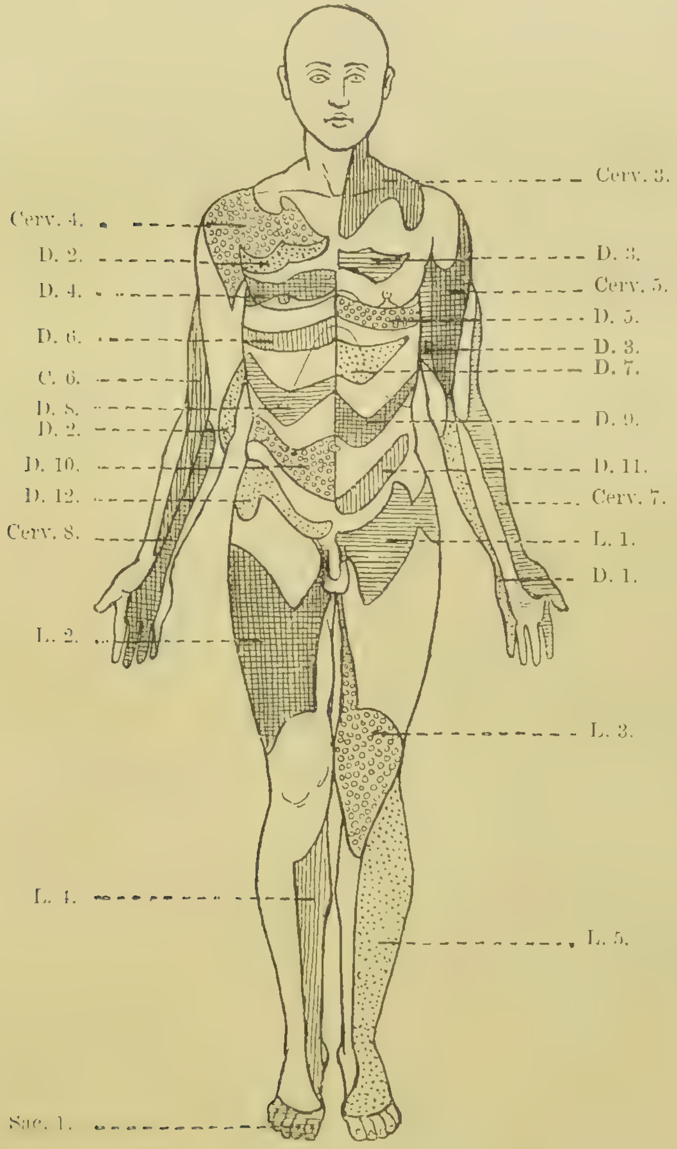

Fig. 161.—Front view of Figure 160.

Head's areas also correspond to the areas affected with attacks of *herpes* (zoster) which is now almost certainly proved to be due to some irritative lesion of the posterior root or root-ganglion, or of the corresponding spinal segment. It was, indeed, the careful study of a large number of cases of herpes, and the careful examination of 21 fatal cases, which enabled Dr. Henry Head to map out these algetic areas with the precision which he has achieved.

TABLE OF VISCERAL DISEASE IN RELATION TO SUPERFICIAL TENDERNESS OR REFLECTED PAIN.[1]

Heart, Ventricle	Dorsal 1 (?), Dorsal 2, 3, 4, 5.
Auricle	Dorsal 5, 6, 7, 8, and (?) 9.
Aorta, Arch	Cervical 3 and 4, Dorsal 1, 2, 3, 4.
Dorsal Aorta	Dorsal 5, 6, 7, 8, 9.
Abdominal Aorta	Dorsal 10, 11, 12, and Lumbar 1.
Lungs	Cervical 3, 4, Dorsal 3, 4, 5, 6, 7, 8, 9.
Œsophagus	Dorsal 5, 6, 7.
Stomach	Dorsal 6, 7, 8, 9, 10.
Intestine	
1. Duodenum to Sigmoid Flexure	Dorsal 10, 11, 12.
2. Rectum	Sacral 2, 3, 4.
Liver and Gall Bladder	Dorsal 7, 8, 9, 10. (Right side).
Kidney and Ureter	Dorsal 10, 11, 12, Lumbar 1, and Occipital Lumbar 2.
Bladder	Sacral 2, 3, 4.
Prostate	Sacral 2, 3, 4, and Occipital Dorsal 10 and (?) 11.
Testicle	Dorsal 10.
Epididymis	Dorsal 11 and 12.
Ovary	Dorsal 10.
Uterine Appendages	Dorsal 10, 11, 12, Lumbar 1, and (?) Lumbar 2.
Cervix Uteri and Lower segment of Uterus	Sacral 2, 3, 4.

Area on the Scalp.	Area on the Trunk.	Organs in relation with these Areas.
Frontonasal.	Cervical 3.	Lung (Apices), Stomach, Liver (Occipital).
	Cervical 4.	
Midorbital.	Dorsal 2.	Heart (Ventricle), Aortic Arch, Lungs.
	Dorsal 3.	
	Dorsal 4.	
Frontotemporal.	Dorsal 5.	Lung, Heart.
	Dorsal 6.	Lungs (lower lobes), Heart (Auricle).
Temporal.	Dorsal 7.	Lungs (lower lobes), Heart (Auricle), Stomach (Cardiac end), Liver (R. side).
Vertical.	Dorsal 8.	Stomach, Liver and Gall Bladder, Lungs.
Parietal.	Dorsal 9.	Stomach (Pyloric end), Liver.
Occipital.	Dorsal 10.	Intestine, Liver, Ovary, Testis, Stomach (Occipital).

§ 552. IV. **Acute transverse myelitis** is a transverse softening of the cord, due to inflammation, thrombosis, or some similar vascular lesion, characterised in typical cases by the completeness of the loss of sensation and motion below the lesion, by the "girdle" pain, and by bedsores and sphincter troubles. The dorsal region is the most usual position of the lesion. This paraplegia *at first* is flaccid.

Symptoms. 1. The advent may be as sudden as apoplexy, or it may occupy a few days. Generally it is attended by slight pyrexia. According to the severity of the onset cases are described as acute or sub-acute. 2. The "girdle" pain is a characteristic symptom, consisting of a feeling as of a constricting cord around the trunk opposite the upper limit of the lesion. There is also a band of hyperaesthesia and increased superficial reflexes in that position. Below this level the sensation and superficial reflexes are lost. 3. Paralysis and anaesthesia involve the whole of both legs. The paralysis is flaccid in the early stage, but the muscles gradually become stiff in the course of a few weeks owing to the descending sclerosis. There is no atrophy and no Erb's reaction, unless the cervical or lumbar enlargements are involved, when the paralysis is of an amyotrophic type (§ 594). The extent of the paralysis, and whether the trunk and arms are involved, depend upon the situation and extent of the lesion. Involuntary startings of the legs are common. 4. The deep reflexes below the lesion are at first abolished, but they rapidly return (except when the lumbar enlargement is involved), then become exaggerated and accompanied by ankle-clonus. 5. Retention of urine and fæces are present at the onset; later on evacuation takes place without the patient being conscious of it. If the lumbar centres are involved there is true incontinence (§ 314) from the onset. Bedsores nearly always supervene, in spite of the best nursing, especially in cases where the lumbar region is involved. 6. The course is always rapid, and there is but little hope of regaining the use of the affected limbs, even partially. In an ordinary case sensation may return in 3 to 6 months, and motion to some extent in 6 to 18 months. The higher up or lower down the lesion is situated in the cord, the more grave the prospect. When the cervical region is affected, death often occurs in a few days from pneumonia, or paralysis of the respiratory muscles. When the lower dorsal region is affected, death may ensue from cystitis, surgical kidney or bedsores.

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[1] Dr. Henry Head in "Quain's Dictionary of Medicine," 3rd Edn., p. 1138.

The *diagnosis* in typical cases of myelitis is not as a rule difficult on account of the girdle pain, rapid involvement of the bladder and rectum, and the completeness of the paralysis and anæsthesia. Compression paraplegia presents pain, is of more gradual onset, and there may be unilateral predominance. Acute myelitis may also have to be diagnosed from hæmorrhage or embolism of the cord (below).

Causes. Acute myelitis is more common in males between 10 and 40 years of age. Among the causes may be mentioned injury, hæmorrhage, extension of inflammation from the meninges, compression, over-exertion, exposure, suppression of the menses, and various toxic blood states. Some cases have been associated with syphilitic disease of the arteries.

Chronic transverse myelitis may supervene on the acute, or it may be chronic from the onset. It presents the same characteristics as acute myelitis in a lesser degree—complete loss of motion and sensation in both legs, girdle pain, involvement of bladder and rectum, and tendency to bedsores. At first before the rigidity supervenes it may resemble multiple neuritis.

Acute diffuse or **central myelitis** is a rare form of acute myelitis accompanied by marked pyrexia, widespread paresis and anæsthesia (§ 568). It rapidly spreads to the whole cord, and is generally fatal.

§ 553. V. Hæmorrhage is said by most observers to be very rare unless preceded by some congenital cystic defect, or some degenerative or neoplastic lesion, such as glioma.[1] The exacerbations of symptoms in syringomyelia are believed to be due to hæmorrhage. Intra-medullary hæmorrhage is characterised by the sudden onset of complete motor and sensory paralysis, accompanied at first by loss of superficial and deep reflexes, which return in an exaggerated degree a few days later unless the lumbar enlargement is affected. It presents most of the symptoms of acute myelitis, from which it can only be differentiated by its instantaneous occurrence, attended sometimes by severe localised pain. Hæmorrhage into the spinal membranes has similar symptoms, accompanied by pain due to pressure on the nerve roots, as in other extra-medullary tumours (§ 550).

VI. EMBOLISM OF THE CORD is rare. The patient complains of a severe "shock" in the spine, followed by sudden and complete paralysis within a definite area which corresponds with the position of the lesion. Cardiac disease or the other causes of embolism are in operation.

§ 554. VII. Spinal pachymeningitis (chronic spinal meningitis, meningeal thickening), may give rise to paraplegia with stiffness of the legs, of very gradual onset and prolonged course. This morbid condition is said by some to be infrequent, and not to give rise to any obvious symptoms, but at the Paddington Infirmary I had no difficulty in collecting 7 cases verified by autopsy in the course of two or three years.

1. The leading *symptom* of this form of paraplegia is the pain shooting down nerves of the legs and elsewhere, aggravated by any movement of the back; sometimes extremely severe, and accompanied by tenderness of the spine. 2. Stiffness and weakness of the legs, involuntary twitchings and increased reflexes were, in my cases, present as the disease progressed. 3. Patches of hyperæsthesia and anæsthesia were occasionally present, and in some there was a progressive atrophic weakness of various muscles due to the constriction of the nerve roots. 4. In certain cases the thecal mischief spreads to the spinal cord, and various symptoms may arise according to the column involved. When the posterior column is affected, as in the cases alluded to in § 573, the patient may present all the symptoms of tabes dorsalis. The course of my cases varied from 2 or 3 to about 10 years, death supervening from some intercurrent malady.

The *causation* is obscure; syphilis was present in the history of some, but none of the cases seemed very amenable to anti-syphilitic treatment.

VIIa. **Cervical pachymeningitis** (hypertrophic cervical meningitis) is the same anatomical condition as the preceding, limited to the cervical region. It was first described by Prof. J. M. Charcot. Pain and atrophic paralysis affect the arms, and subsequently spastic paraplegia due to descending lateral sclerosis.

§ 555. VIII. Chronic system lesions. Three degenerative diseases of the central nervous system of fairly common occurrence may first come under notice for weakness of the legs—disseminated sclerosis, general paralysis of the insane, and occasionally tabes dorsalis. Each have other and more characteristic symptoms, and will be dealt with elsewhere. There are also 5 other somewhat rare conditions in which stiffness of the legs due to lateral sclerosis is a leading feature primary lateral sclerosis (lateral sclerosis only), ataxic paraplegia (lateral and posterior sclerosis), amyotrophic lateral sclerosis (sclerosis of the lateral column and anterior horns), infantile cerebral and spinal paraplegia and toxic sclerosis. (See fig. p. 891).

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[1] See an interesting clinical lecture by Sir William Gowers in *The Lancet*, 1903, vol. ii., p. 993.

IX. **Primary spastic paraplegia** (syns. primary lateral sclerosis, primary sclerosis of the crossed pyramidal tracts, Erb's paraplegia,[1] tabes dorsalis spasmodique). Lateral sclerosis on one or both sides is commonly met with as spreading downwards from a localised disease in the brain or cord, but the primary spastic paraplegia consists of sclerosis of these columns without any primary disease above. Some deny the existence of a primary lateral sclerosis, and personally I regard the disease as extremely rare, though some cases of spastic paraplegia seem to baffle all inquiries as to the primary or initiating lesion. (1) The onset is extremely slow and insidious, and the course is slow and painless, extending over many years, 20 or 30 being said to be not uncommon. (2) Dull aching and stiffness rather than absolute weakness is the leading symptom, and this gradually results in a stilted walk, till finally the patient walks on tiptoe without bending the knees, which is very characteristic. In advanced cases there is cross-legged progression because the adductors are specially involved. Both legs are involved, though one may be a little worse than the other. Increased knee-jerk and ankle-clonus are present as in other cases of lateral sclerosis. (3) The other symptoms consist of a series of negative points, viz., no disturbance of sensation, no sphincter trouble, no bedsores, and no alteration in the electrical reactions are the rule. In the later stages of the case the arms may become stiff.

Etiology. The patients are usually from 30 to 40 years of age. Deficient blood supply has been suggested, and syphilitic toxæmia leading to disease of the arteries is a possible cause. Chronic alcoholism and other toxic agencies have been suggested.

X. **Ataxic paraplegia,** or postero-external sclerosis, is a rare disease due to primary sclerosis in the posterior as well as the lateral columns. There is defective co-ordination of the movements and ataxy, but none of the other symptoms belonging to locomotor ataxy. The knee-jerks are increased and there is stiffness of the legs. This disease is met with chiefly in men of middle age, but much the same spinal lesions occur in Friedreich's disease, *i.e.*, the hereditary ataxy of childhood.

XI. **Amyotrophic lateral sclerosis** (Charcot) is a rare condition due to disease affecting both the anterior horns and the lateral columns. The symptoms are usually confined to the arms for a time, and the disease is therefore described under brachiplegia, § 562.

XII. **Infantile cerebral and spinal paralyses** (including birth palsy; spastic diplegia; Little's disease). The cerebral leg centres lie on the mesial aspect just below the margin of each hemisphere, and during difficult or prolonged labour these are liable to damage by compression of the hemispheres and meningeal hæmorrhage. In other cases diffuse atrophy has been found, or porencephalus. When the condition occurs in prematurely born infants it is believed to be due to defective development of the pyramidal tracts. Consequent on any of these lesions degenerative sclerosis takes place down the crossed pyramidal tracts, and spastic paraplegia results. Nothing perhaps is noticed until the child begins or ought to begin to walk, which it is late in doing; then the legs are stiff, and signs of lateral sclerosis are present. The arms also may be involved—spastic diplegia. A limited athetosis or a more generalised choreaic spastica may ensue. In all of these varieties there is very often a history of convulsions in infancy and a condition of mental deficiency. In other cases (hereditary spinal spastic paraplegia), however, the child is born with a stiffness of the legs but without any mental symptoms or backwardness; and in these cases there has possibly been an injury to the cord at birth. Such patients may live bedridden to a considerable age, 40 or 50 years. Some recognise a genuine hereditary form of the malady which affects several members of a family, in whom some developmental defects may be assumed to exist.

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[1] Erb has described under the term syphilitic spinal paralysis an insidious spastic paraplegia, which he regards as distinct from primary spastic paraplegia, but in the author's view this is practically identical with the above. Trans. West Lond. Med. Chir. Soc. about 1902.

XIII. **Toxic combined scleroses.** Of late years sclerosis of the cord has been associated by the labours of Taylor,[1] Russell, and others with various blood conditions, such as diabetes, profound anæmia, leukæmia, and pellagra. The paralysis is never very great. The symptoms vary according to which of the columns is mainly affected, but paraplegia is generally present in some degree, and is sometimes associated with ataxy and numbness. The sclerosis affects chiefly the posterior and lateral columns of the cervical and thoracic regions. It may be recognised from similar spinal affections by the previous occurrence of one of the diseases named or some other toxic disorder, anæmia being perhaps the most typical instance.

Progressive stiffness of the legs resulting in spastic paraplegia is the leading symptom of **lathyrism** (lupinosis), *i.e.*, poisoning by the use of meal derived from the seeds of the chick-pea (lathyrus satirus and l. cicera) when mixed with other meal in the preparation of bread. The arms are very rarely affected. It is met with in India (chiefly), Algeria, and probably elsewhere. The anatomical condition is not known, but it is probably a toxic sclerosis.

(b) **The lower neuron or flaccid paraplegias** constitute a more limited group than the upper neuron paraplegias, and are distinguished from them by the four following features:—(1) the paralysis is flaccid, at any rate for a considerable time; (2) muscular wasting is a marked feature, and is attended by (3) characteristic electrical changes; (4) the deep-seated reflexes are absent. This group is distinguished from the functional paraplegias (Group c) by the steady and progressive course of the lower neuron paraplegias, by the muscular wasting, and the electrical changes.

I.	Multiple Peripheral Neuritis	§ 556
Ia.	Beri-Beri	§ 557
II.	Anterior Poliomyelitis	§ 595
III.	Acute Transverse Myelitis (at the outset)	§ 552
IV.	Landry's paralysis	§ 570
V.	Syringomyelia and intra-medullary tumours, intra-medullary hæmorrhage and embolism (sometimes)	§ 601

§ 556. I. **Multiple peripheral neuritis** (syn. multiple neuritis; polyneuritis) is a symmetrical inflammation of the peripheral nerve trunks (due to a toxæmia) attended with pain and tenderness along their course, resulting in paresis and anæsthesia. This disease is more or less general in its distribution, but the paresis may predominate in the legs, and the patient frequently comes under our notice for paraplegia.

Symptoms. The onset may be acute, sub-acute, or (more usually) chronic. Some observers make 3 varieties according to whether the motor, sensory, or ataxic symptoms predominate. In acute cases pyrexia may be present. (1) There is generally a premonitory stage in which there is numbness, tingling, cramps and twitchings in the legs, or arms. Pain forms a prominent feature in all cases of neuritis, the degree varying according to the acuteness of the process. The pain shoots along the course of the nerves, is symmetrical in distribution and increased by movement. (2) There is also deep-seated tenderness along the nerve trunks and in the muscle substance, especially in the calves. These symptoms are soon followed by (3) flaccid paresis attended by more or less atrophy. The extensors are more affected than the flexors, and the patient has a characteristic foot-drop (or wrist-drop) and a difficulty in raising the legs in mounting stairs. (4) The electrical reactions vary, but there is in all cases a diminution of faradic reaction. In many cases there is also a diminution to both galvanic poles (a condition which one does not obtain in anterior polio-myelitis), while in a few cases there is also typical Erb's reaction (§ 516), and A. C. C. is greater than K. C. C. (5) The superficial and deep reflexes are diminished or disappear. (6) Anæsthetic and hyperæsthetic areas at the termination of the sensory nerves in the legs and arms may, with care, be revealed. Often there is a patch of anæsthesia surrounded by a zone of hyperæsthesia. Bladder and rectal troubles and bedsores are only met with in very severe and extensive cases, though in alcoholic cases with the dull mental state the excretions may be passed in bed. It is an interesting fact that the nerves involved vary somewhat with the toxic agent in operation, as though the latter had some selective proclivity. Thus alcohol and beri-beri affect mainly the legs; lead, the arms; diphtheria, the throat and neck muscles; while arsenic appears in a few cases to affect mainly the trophic nerves of the skin. In lead paralysis the toxin selects the motor fibres of the musculo-spiral nerve and spares the sensory. Accompanying severe alcoholic cases is loss of memory, apathy, and sometimes delirium.

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[1] James Taylor, Roy. Med. Chir. Soc. Trans., 1895; J. Risien Russell, *The Lancet*, 1898.

Peripheral neuritis may have to be *diagnosed* from tabes and poliomyelitis. It is recognised from the former by the actual muscular weakness present, the high-stepping walk, and the pupils and from the latter by the sensory changes and the age of the patient. The neuralgic pains may have to be differentiated from other causes of "pains in the limbs" (§ 423); and lastly the disease will often need to be distinguished from the other flaccid lower neuron pareses in this group, and from syringomyelia

(§ 601). The acute febrile cases are hard to distinguish from Landry's paralysis and poliomyelitis in adults (§ 568).

Prognosis. Multiple neuritis is essentially a chronic affection but has a distinct tendency to recover. The chief danger rests in the involvement of certain nerves, such as the pneumogastric in diphtheritic paralysis, when sudden death may ensue. The acute febrile cases may die in a week from involvement of the respiratory muscles or cardiac paralysis, but in general terms a large proportion of cases of multiple neuritis get well under appropriate treatment in the course of 3 to about 12 months, if the cause in operation be removed. Deformities from contractures may result in severe cases.

Causes. Multiple neuritis may occur in either sex and at any age, but it is most common in adults from 20 to 50. Women are slightly more affected than men. There is always some toxæmia or blood condition in operation; and the commonest of these in adults is alcohol, in children diphtheria. The other causes are, among the heterotoxins—lead, arsenic, silver, mercury, ether, bisulphide of carbon and naphtha; among the microbic toxins—tuberculosis, leprosy, beri-beri, enteric fever, variola, (?) rheumatic fever, scarlatina, and other infectious fevers. Peripheral neuritis may supervene in some cachectic or blood conditions, *e.g.*, cancer, gout, diabetes, and severe anæmia; and an attack has sometimes followed over-exertion or exposure. Traumatic causes come under the head of monoplegias.

Treatment. The causes must be sought for and removed, *e.g.*, alcohol must be absolutely forbidden, and any gouty or other diathesis must be treated. Rest is the cardinal feature in all treatment, and merely remaining in bed will do much to cure. Iodide of potassium and quinine are undoubtedly of use for the elimination of toxin, whether it be alcohol, lead, gout, or other kind. When the acute and painful stage has subsided galvanism is very valuable, and its systematic use, with or without faradism and massage, will hasten the restoration of the muscular weakness.

§ 557. Ia. Beri-beri frequently comes under notice for paraplegia, sometimes for dropsy of the legs. The disease is endemic or epidemic in certain localities. The paraplegic form is now recognised as due to a peripheral neuritis.

The *symptoms* of beri-beri belong to two classes, (a) those referable to the neuro-muscular system (the paralytic type); and (b) those referable to the vaso-motor system (the œdematous type). Either may occur alone, but more often both are present, the paralysis predominating. The onset may be sudden or gradual, with prodromata of languor, pains, and slight dyspnœa, In (a) there is no œdema. The patient may be so thin and emaciated that the condition is called "dry beri-beri." The first symptom is a difficulty in walking, with the characteristic high-stepping gait of foot-drop. The knee-jerk is soon lost, and the muscular paralysis may extend from the legs to the arms, diaphragm, intercostals and larynx. There is cutaneous anæsthesia, occurring first in the legs, and sometimes not spreading further; at the same time there is great muscular tenderness, especially noticeable in the calves. The sphincters are not affected. The heart is dilated towards the right.

In (b) the characteristic symptoms are great dyspnœa and œdema, which is epecially seen in the legs. Where dropsy is extreme the condition is called "wet beri-beri," the heart is dilated, especially to the right, with changing bruits from day to day. The digestion may remain unimpaired, and there is no fever.

Diagnosis. Beri-beri has to be diagnosed from alcoholic and other forms of peripheral neuritis, and here the œdema, and the constant involvement of the heart in beri-beri are important. It may be diagnosed from locomotor ataxy by the absence of the characteristic ocular and other symptoms which occur with that affection.

Prognosis. In an epidemic the mortality may rise to 50 per cent.; but normally only 10 per cent. of the cases end fatally. Death occurs usually from cardiac failure or asphyxia, and is thus very frequently sudden and unexpected. A favourable case runs its course in a few weeks, but usually the disease lingers on for months, especially if the patient continue to live in an endemic area. Serious complications may arise in the form of œdema of the lungs or effusion into the serous cavities.

Causes. Beri-beri occurs in Japan, China, the Malay Peninsula, parts of America, the West Indies, East Africa, and less often in India. It attacks all ages, but is less frequent in children and old people. Beri-beri occurs in outbreaks year after year in the same place; if it has once occurred in a ship it is very difficult to disinfect that ship so that the disease will not arise in it again and again. If one case occurs in a crowded prison or town the disease will rapidly spread over the whole community. It is not known how the malady is conveyed.

Treatment. When one case occurs in a crowded school or prison everyone should be sent away immediately. If a case occurs on board ship the patient must be brought on deck, and kept in the fresh air, and on reaching land the ship must be thoroughly disinfected. When a patient acquires the disease on land he must be immediately removed to another locality and isolated. As regards diet, all bulky food (and therefore rice) must be avoided. Other treatment is symptomatic, and if symptoms of great venous obstruction occur with intense dyspnœa, bleeding must be at once resorted to.

II. **Acute anterior poliomyelitis** at the onset may take the form of a paraplegia, but it more often affects one of the legs, or one of the arms. In the course of a week or two, it always settles down into one limb, one segment of a limb, or one set of muscles. It is almost entirely confined to children. It is described under amyotrophy (§ 595).

III. **Acute transverse myelitis** (§ 552) is at the outset a flaccid paraplegia, but the paralysis is not atrophic excepting in the unusual event of the lesion occupying the cervical or the lumbar enlargement. The paralysed limbs become rigid and the reflexes return in the course of a few weeks.

IV. **Landry's paralysis** is also a flaccid paralysis (§ 570). It is a rare disease resembling acute myelitis in some respects, only no gross lesions after death have been found. There is a sudden onset of flaccid paralysis of the legs followed by rapid extension to the muscles of the trunk and arms, and generally a rapidly fatal termination. A microbe having the form of a tetracoccus has been described in connection with the disease (E. F. Buzzard) but its etiological significance has not yet (1904) been established.

V. **Syringomyelia** and **intra-medullary tumours** and **hæmorrhage** may come under notice as paraplegia with flaccidity, and very often with a contradictory increase in the knee-jerks; but on further examination this will be found to be preceded by the arm symptoms (§ 601).

(c) **Functional** or **variable paraplegias.** When an organic lesion is present, whether it be in the upper or lower neuron, we get a continuous or rather an unvarying paralysis which progresses steadily for better or worse. But the leading feature of functional paraplegia is (1) that it varies in intensity and sometimes in its other features from day to day. (2) The paralysis is less marked, it is a paresis rather than a paralysis, and it does not conform either to the upper or lower neuron type; (3) it is accompanied by other evidences of the causal condition.

The principal varieties of variable paraplegia are:—

I. Hysterical paraplegia.
II. Railway spine.
III. Paraplegia after fevers.
IV. Diver's paralysis.
V. Reflex paralysis.

§ 558. I. **Hysterical Paraplegia** is undoubtedly the commonest and may be taken as the type of functional paraplegias. The nature of the lesion we do not know, and so variable are its clinical features that we have to rely mainly upon the fact of its occurrence in a female who is the subject of other hysterical manifestations. (1) The paraplegia is generally of sudden onset and not infrequently dates from some hysterical seizure. It is rarely complete, and varies in intensity from day to day. Sometimes it is rigid, but almost as often flaccid, and the patient drags her feet along in a characteristic way. It resembles peripheral neuritis in the presence of foot-drop. (2) The paralysis and the other symptoms are inconsistent and evanescent. Thus, there may be hyperæsthesia in one leg, anæsthesia in the other, and the two may change places or disappear from day to day. All the symptoms may disappear rapidly; though I have observed rare cases of several years' duration.[1] (3) The muscles do not waste nor lose their electrical contractility. The deep reflexes may be unchanged, but I have generally found them exaggerated. The plantar reflex gives a normal flexor response showing the absence of organic disease of the spinal cord. (4) Incontinence of urine or fæces are very rare, and bedsores never occur with good nursing. (5) Other evidences of the hysterical diathesis are present (§ 519).

In the *diagnosis* it is well to remember that the patient is practically always a female, though male cases have been recorded.[2] In myelitis there are trophic changes, girdle pain and incontinence, features which are wanting in the functional variety. Other organic paraplegias are identified by their continuous and progressive character and by the presence of ankle-clonus and Babinski's sign. These latter are regarded as absolutely conclusive of organic disease, but there is a kind of spurious ankle-clonus in many cases of hysteria which is only with difficulty distinguished from the ankle-clonus of organic disease. The *causes* and *treatment* are dealt with elsewhere.

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[1] Trans. Clin. Soc. Lond., vol. xxii., and *The Lancet*, vol. i., 1901.
[2] *The Lancet*, vol. i., 1889, and vol. ii., 1889, p. 792.

§ 559. II. **Railway spine.** A severe shock or injury to the spine, such as occurs in railway accidents, may cause (1) a direct injury to the cord such as hæmorrhage, laceration or concussion ; (2) the shock may determine the occurrence of some degenerative lesion which does not come on perhaps for some months, such as disseminated sclerosis, tabes, or spastic paraplegia ; or (3) a form of painful paraplegia may ensue not coming on until some days, weeks, or even months after the injury. It is with this last we are now concerned. Some, like Erichsen and Gowers, regard it an incipient myelitis ; others, like Herbert Page, maintain that it is due to a functional change (? vascular). The symptoms consist of—(1) Severe spinal pain and tenderness (spinal neuralgia), and obscure peripheral sensations such as tinglings, twitchings, or numbness, in the legs. (2) A paresis or pseudo-paralysis, which Page believes may be due to the fear of pain produced by movement. (3) Bladder symptoms are sometimes present, such as frequency of micturition or a dribbling at the end ; or a difficulty in defæcation. These cases are very difficult to treat successfully. Rest with change of scene and occupation are the main elements.

III. **Paraplegia after fevers.** Anæmia and congestion of the cord are said by some to give rise to no symptoms, but by others are said to cause tingling and cramps, especially at night, and a variable degree of weakness of the legs, chiefly on exertion. General anæmia of a severe type has been shown to cause actual sclerosis of the cord (see toxic sclerosis). The paraplegia following beri-beri and most fevers is due to peripheral neuritis, sclerosis (*vide* § 555) or myelitis, but that which is especially liable to occur after jungle fever is held by Indian authorities to be due to anæmia of the cord. All these forms are characterised mainly by (1) the circumstances under which it occurs, (2) the paralysis being incomplete and usually transient.

§ 560. IV. **Diver's Paralysis** (syn. Caisson disease, compressed air illness) is a paraplegia which occurs in men who work under water, or tunnel under the ground, at high atmospheric pressures. It is due undoubtedly not so much to the pressure they are subjected to, as to the sudden release of that pressure, which produces congestion of the cord, with liberation of gas from the blood. In fatal cases hæmorrhage and myelitis have been found. This was well illustrated in a case shown by Dr. Robert Maguire at the Medical Society[1]; the patient had had 6 or 7 previous attacks, and the last was brought on entirely by his sudden rush to the surface from a depth of 150 feet, that is to say, from a pressure of 90 lbs. to the square inch to one of 15 lbs. to the square inch. The paresis is never quite complete, but is of sudden onset when the patient returns to the normal atmospheric pressure. It usually affects the legs, rarely the arms to any extent. Anæsthesia, severe pain and sphincter paralysis only occur in the graver cases. Auditory vertigo, hæmorrhage from the nose, lungs and other parts sometimes occur. The prognosis is favourable in most cases ; pain and paresis pass off in a few days to 6 weeks. A few cases have died.

Treatment. Curative measures consist simply of rest and the avoidance of alcohol, a failing to which most of these workmen are addicted. Ergot had been recommended. Preventive treatment consists in following out precautions for gradual decompression. Men employed in diving or in tunnel working under pressure should be compelled to undergo a very gradual process of decompression. Dr. Snell[2] recommends 10 minutes decompression for each atmosphere of pressure.

V. **Reflex paraplegia** is a variety which some do not admit, but there appear to be certain very rare cases associated with gastro-intestinal, uterine, or other irritation. It was first described by Trousseau. After operations on the anus there may be weakness of the legs and inability to pass urine for days. The loss of power appears to be never quite complete. The only means of identification consists in the presence of a reflex cause, and the disappearance of the paralysis when this is removed. Some cases supposed to be of reflex origin have been found to be due to neuritis ascending to the cord.

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[1] *The Lancet,* April 14, 1900.

[2] "Monograph on Caisson Disease," by Dr. E. H. Snell. H. K. Lewis, London, 1896.

§ **561.** *General remarks on the prognosis of paraplegia.* Paraplegia is always a serious symptom, not only because it prevents locomotion but because it indicates structural or functional disease of that important structure the spinal cord. In general terms (1) all functional disorders are more favourable than organic; (2) flaccid paraplegias tend to run a quicker course either towards recovery or death than rigid paraplegias: (3) the most unfavourable signs are bedsores, and the implication of the bladder and rectum. The different forms of paraplegia may be grouped for the purposes of prognosis into four groups (see table below). The presence of complications adds to the gravity of any case. Many cases die of cystitis and pyelitis, or the chronic septicaemia which results from bedsores; others from pneumonia or other pulmonary complications. Finally, the prospect of efficient or inefficient nursing is an extremely important factor in the prognosis, for these cases constitute the test and trial of all that makes for efficiency in true nursing.

Prognosis of the different forms of paraplegia.

Those which generally recover completely either spontaneously or under adequate treatment.	Those which generally terminate in partial recovery either spontaneously or under adequate treatment.	Those which tend to become chronic and incurable, but not fatal.	Those which tend to a fatal issue.
Hysterical and the other varieties of functional paraplegia. Peripheral neuritis. Some cases of Pott's disease. Non-malignant extra-medullary tumours. Paraplegia after fevers. Toxic sclerosis.	Most cases of Pott's disease. Anterior poliomyelitis.	Dorsal myelitis. Embolism. Some spinal injuries. Chronic spinal meningitis. Hæmorrhage. Primary lateral sclerosis. Ataxic paraplegia. Amyotrophic lateral sclerosis. Infantile cerebral and spinal paraplegia.	Malignant and inaccessible tumours. Acute cervical or lumbar myelitis. Some cases of hæmorrhage. Malformations.

The *treatment of paraplegia*, excepting in "functional" cases, is not very hopeful, for a damaged portion of the cord can never be fully restored. There are three indications—to remove the cause, to prevent complications, and to restore the functions. (*a*) To remove the cause—(1) anti-syphilitic treatment, and particularly iodide of potassium should be employed at once in all possibly syphilitic cases; 60 to 180 grains of the iodide should be given daily. It will probably not restore a damaged segment of cord, though Brown Séquard advocated its use in what were called cases of atrophic softening. In some chronic cases it certainly seems to promote the absorption of inflammatory products. (2) If the existence of inflammation or congestion of the cord be suspected, belladonna and ergot may be given: experimentally these remedies produce contraction of the vessels of the pia mater. Rest and the prone position, the patient lying on the stomach, are also of advantage. In cases of congestion or myelitis, nux vomica, opiates, and galvanism should be avoided. Where hæmorrhage is suspected prolonged absolute rest in the prone position is necessary. (3) Counter-irritation I believe might with advantage be used more often than it is, especially in the upper neuron group—blisters to the spine, frequent cupping, hot douches (98° to 101°) frictions, mustard or ung. hyd. iod. rubbed into the spine. In chronic cases, the best treatment, in my experience, is the "coup de fer" of the French, that is to say, dotting the point of a Paquelin thermo-cautery down the spine every other day. Several cases of sclerosis under my care have derived advantage from these methods. (4) Surgery comes to our aid in cases of Pott's disease, injury, malformations, and some tumours. Certain of the causes require special treatment. In *Pott's disease* rest and extension in a prone or supine position afford a good chance of complete recovery (head and arms fixed to the wall, counter extension by weight). In older people laminectomy may be done at once because the disease tends in them to progress. Various methods of forcibly reducing dislocation are now adopted. Sayre's jacket and various kinds of apparatus are used; one of the best of these perhaps is one designed by Mr. Jackson Clarke.[1] *In acute myelitis* an ice-bag to the spine, and the administration of atropine subcutaneously and locally have been advocated, though but little can be done beyond rest and the prevention of bedsores. For *chronic meningitis* (pachymeningitis) and the six upper neuron scleroses (VII. to XII.) the iodides or atropine may be tried, but counter-irritation is the best. The treatment of *hysterical paraplegia* is given elsewhere, but the value of faradic and static electricity is undoubted.

(b) To prevent complications should be our endeavour in all cases. A catheter carefully *asepticised* should be passed 3 or 4 times in the 24 hours in cases of partial or total retention, and with considerable care, for the parts are often anæsthetic. The patient should be placed upon a water bed; the bowels kept gently acting with laxatives or enemata; and the formation of bedsores prevented by cleanliness, dryness, and the relief of pressure at all prominent points. The question of nursing is of the highest importance in all of these cases, especially in cases of myelitis.

(c) The restoration of the function of the muscles may be promoted in due course by the application of galvanism, faradism (particularly the combined current), massage, and passive movements. These means are more useful in flaccid paraplegias, and are not suitable in recent or irritable cases, or in the earlier stages of acute myelitis. Nervine tonics, strychnine, phosphorus, arsenic, cod-liver oil, iron, quinine, are all of value to promote nutrition. Bearing in mind the view of modern pathologists, that in cases of sclerosis of the cord, the primary lesion is a malnutrition of the nerve-cells and fibres, rest and a liberal dietary, especially one containing abundance of cream and fats, may be of use, and in actual practice I have found it so.

The patient complains of weakness or **paralysis** *of* BOTH ARMS —the case is one of BRACHIPLEGIA.

§ 562. **Brachiplegia** is paralysis of both arms without paralysis of the legs. It is not a very common condition and must not be confused with diplegia, which is double hemiplegia, nor with brachial monoplegia. It is met with occasionally in the following diseases. In the *diagnosis of the cause* you should first ascertain whether you have to do with a *lower* neuron lesion such as 1, 2, 3, or *upper* neuron lesion as in 6 and 7.

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[1] See discussion Clin. Soc., *The Lancet*, March, 1900.

(1) Double *musculo-spiral paralysis* (§ 564) is the commonest cause of brachiplegia, as met with in lead poisoning and sometimes other toxic conditions such as poisoning by arsenic, or silver.

(2) In *syringomyelia* (§ 601) wasting and weakness of one or both arms may be the earliest feature. The paralysis is usually flaccid, but may be rigid, and is accompanied by loss of temperature sense and other sensory changes.

(3) In a few cases of *acute anterior poliomyelitis* (§ 595) both arms may be paralysed at the same time.

(4) In certain cases of *idiopathic muscular atrophy* weakness of the muscles of the arms and shoulder girdle may be the earliest and for a long while the only symptom.

(5) *Injury to the cord* in the cervical region may result in atrophic paralysis of the arms.

(6) In *hypertrophic cervical meningitis*, and in *extra-medullary tumour* in the region of the cervical enlargement, there is acute pain in the arms, and the "claw hand" usually develops.

(7) Amyotrophic lateral sclerosis (Charcot) is a rare disease, due to disease of the anterior horns and also of the lateral columns. In the first stage, lasting usually from 4 to 12 months, the symptoms are mostly confined to the upper extremities, which are feeble and gradually *undergo atrophy*. Combined with this is rigidity and increase of the deep reflexes of the arms, which gradually assume a characteristic position owing to the contracture of the muscles. Both arms are usually involved, but not infrequently one side predominates. In the second stage the lower extremities become invaded by the symptoms already described under primary lateral sclerosis. The disease differs from progressive muscular atrophy, of which some regard it as a variety, in the presence of the stiffness both of the arms and of the legs, and also in its relatively rapid course, for death generally takes place in from 1 to 3 years. It more closely resembles cervical pachymeningitis, excepting in the absence of pain.

The patient complains of weakness or paralysis *in* ONE ARM *or* ONE LEG—the case is one of MONOPLEGIA.

§ **563. Monoplegia** is loss of power in one limb. Monoplegia brachialis is paralysis of one arm; monoplegia cruralis, of one leg.

The chief POINTS TO INVESTIGATE are—1st, precisely which of the muscles is affected and the character of the paralysis; 2ndly, the electrical reaction; 3rdly, the sensation of the affected part; and 4thly, the presence of any cause of pressure existing along the course of the nerve trunks, plexuses, or roots. It is often difficult to decide whether an alleged weakness in the forearm is not in reality due to disease in one of the smaller joints. In any case the joints should be examined because even after slight injury an arthritic amyotrophy (see below) occasionally ensues which may cause weakness in the limb.

Monoplegia may be due to a lesion situated (a) in the peripheral nerves, plexuses, or roots; (b) spinal affections involving the anterior horns, or anterior roots; or very rarely (c) localised cerebral lesions; the first two being paralysis of the lower neuron, the third those of the upper neuron type.

a. Single nerve and plexus paralysis (§ 564).

I. Single nerve paralysis.
II. Plexus Paralysis.
III. Occupation neuroses.
IV. Arthritic atrophy.

b. Spinal monoplegias (§ 566).

I. Acute anterior poliomyelitis.
II. Chronic anterior poliomyelitis.
III. Spinal tumours, Pott's disease, and other causes of paraplegia (occasionally).
IV. Syringomyelia.
V. Amyotrophic lateral sclerosis, cervical pachymeningitis, and other causes of brachiplegia (occasionally).

c. Cerebral monoplegias (§ 567).

I. Focal cortical lesions.
II. Hysterical monoplegia.

d. Certain primitive myopathies (§ 597).

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§ 564. I. **Single nerve paralysis.** Peripheral nerve and nerve-plexus lesions give rise to a monoplegia which, like all lower motor neuron lesions, is flaccid, accompanied by R. D., and is followed by atrophy of the affected muscles. The key to the nerve or nerves involved consists of the identification of the precise muscles affected. The lesion may be an injury, pressure, or inflammation, but the symptoms are much the same in kind though they differ somewhat in degree. A divided nerve may be taken as the type.

Symptoms. Division of a *mixed* nerve (or its severe contusion or compression) gives rise at once to (1) flaccid paralysis of the muscles supplied, (2) alterations in the electrical reactions, (3) abolition of the tendon reflexes in the muscular region involved, followed in the course of the ensuing week by (4) muscular atrophy. (5) Pain and various kinds of paræsthesia are very constant; indeed, pain is the most prominent symptom in acute inflammation or severe injury. (6) Anæsthesia is much less constant. Sensation may remain intact even after complete section owing, it is said, to the extensive anastomoses of the nerves. Sensation when lost is sooner recovered than motion. Persistent and extensive anæsthesia indicates destruction or section of more than one nerve trunk or of a whole plexus. (7) Vaso-motor and trophic disorders often result—redness, hyperidrosis, œdema, wasting of

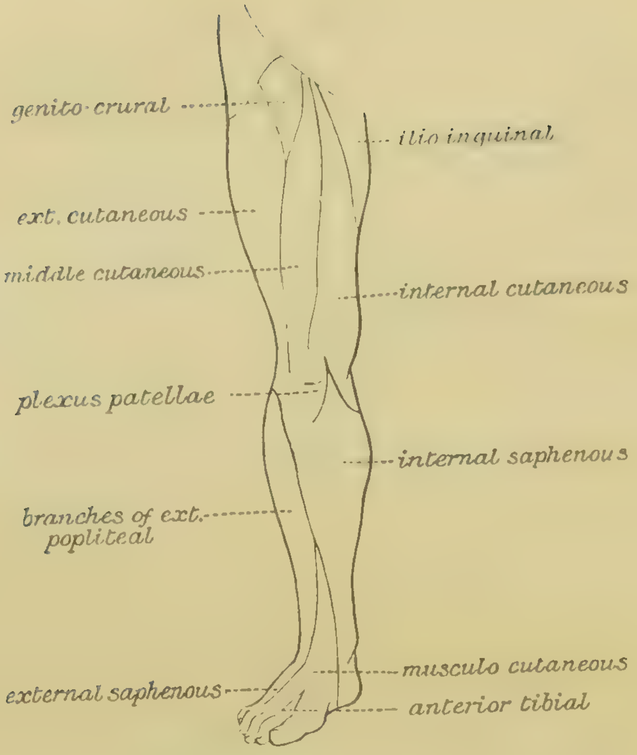

Fig. 162.—Approximate areas of CUTANEOUS SENSATION supplied by the peripheral nerves.

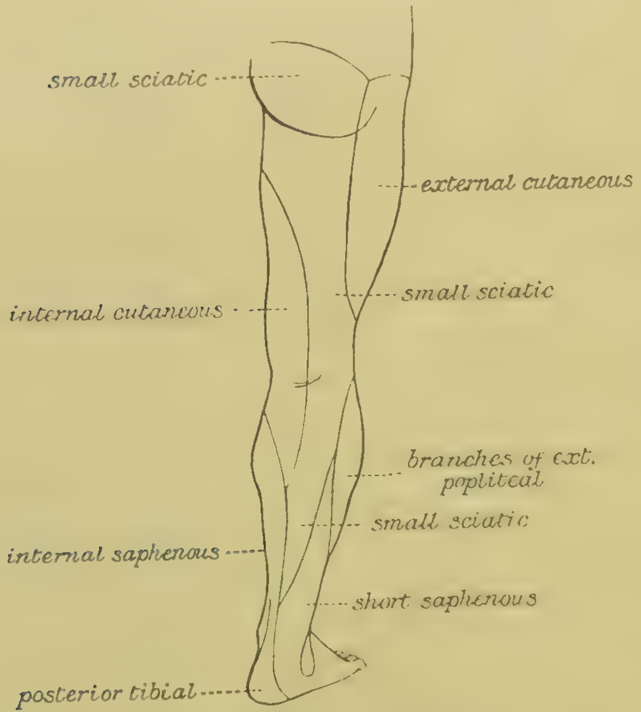

Fig. 163.—Approximate areas of CUTANEOUS SENSATION supplied by the peripheral nerves.

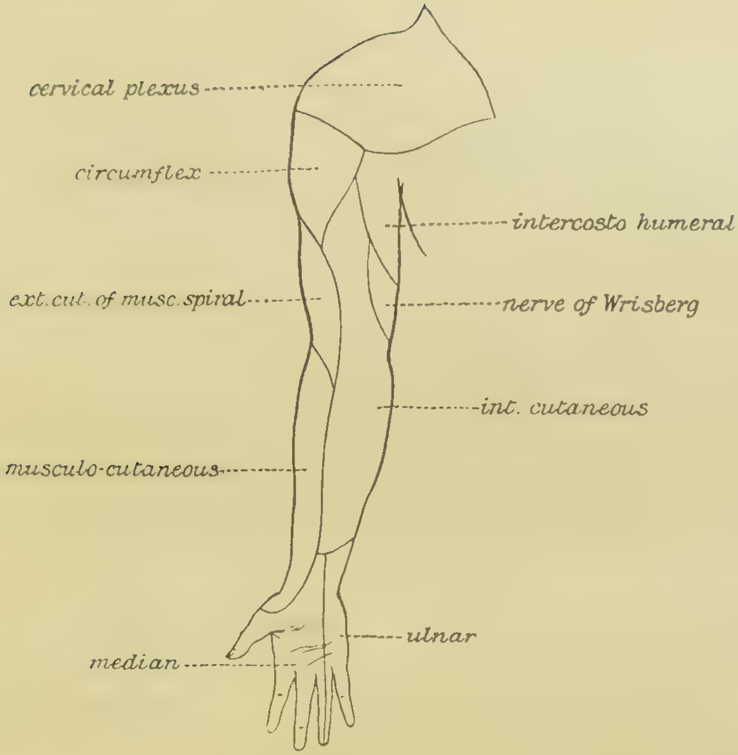

Fig. 164.—Approximate areas of CUTANEOUS SENSATION supplied by the peripheral nerves.

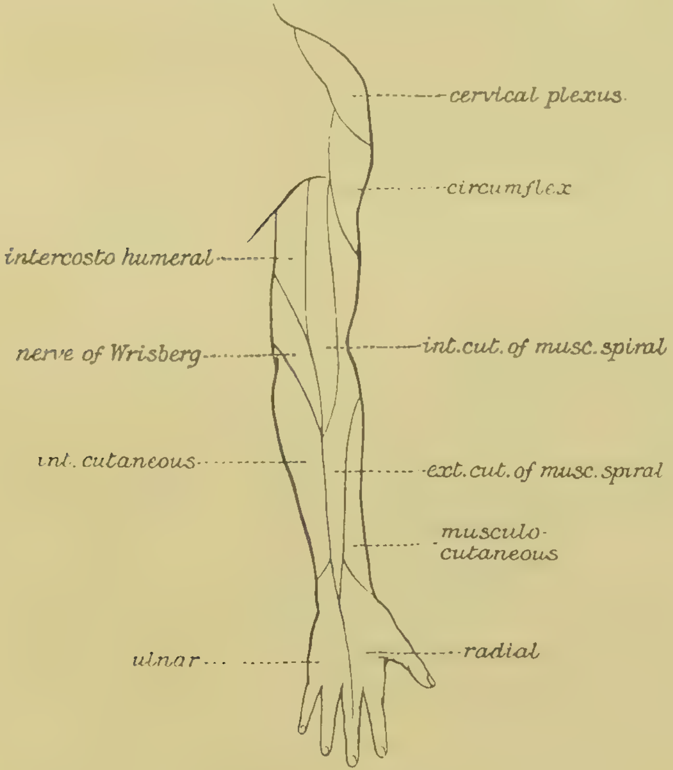

Fig. 165.—Approximate areas of CUTANEOUS SENSATION supplied by the peripheral nerves.

the skin (glossy skin) subcutaneous tissue and bones, and vesicles followed by badly healing sores.

The *diagnosis* may always be accomplished by the electrical reaction of degeneration. Thus we are enabled to distinguish a nerve injury from (1) a direct muscular injury, (2) arthritic atrophy following a slight contusion of the joint. (3) Spinal monoplegias are distinguished by the different course they run and the associated symptoms (p. 1017).

The *prognosis* depends to some extent upon the degree and cause of the injury on compression. Complete R. D. indicates complete severance or destruction of a nerve. But it is wonderful how a nerve will repair with rest, as Mr. John Hilton[1] pointed out long ago.

The *causes* of peripheral nerve and plexus lesions are manifold but may be grouped under two headings as exemplified mainly in the musculo-spiral nerve. The special causes affecting the several nerves will be considered afterwards.

a. Injury or *pressure.* Pressure during sleep (especially after alcoholic intoxication), ligatures, the use of crutches (crutch-palsy of the musculo-spiral), luxation of the humerus or other bones, bullet wounds, stabs and direct blows on a nerve or nerve trunk (*e.g.*, brachial plexus above the clavicle), or fractures, may injure the nerve; later, callus (recognised as a cause by the pain coming on some time after the injury) may involve or compress a nerve, or the nerve may be included in a cicatrix (internal or superficial). Sudden extension of the arm upwards may lead to severe damage or laceration of the brachial plexus; damage during parturition may act on these or other nerves and severe muscular action (*e.g.*, contraction of the triceps on the musculo-spiral) may act (as Sir William Gowers has shown) by compression. Diseases of the bones beside a nerve or through which a nerve passes (*e.g.*, caries or syphilitic disease), or enlarged glands or other tumours may lead to compression, and extensive pleurisy at one apex may have the same effect. A small injury of the thumb may lead to ascending neuritis (Kausch, quoted by Oppenheim[2]). Forced functioning leads to occupation neurosis which may result in spasm (*q.v.*), paralysis or tremor.

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[1] Hilton's "Rest and Pain," edited by Jacobson. London, Bell & Sons, 1887. See also a case reported by the author in *Brain*, part lxiii., 1893.

[2] "Diseases of the Nervous System." Lippincott, London, 1904.

β. A *toxic agent* (as already mentioned in multiple neuritis) sometimes acts as a predisposing factor, sometimes as the sole cause; and the toxic agent often seems to have an unexplained proclivity for certain nerves, as in the case of lead for the musculo-spiral. Alcohol generally produces multiple neuritis, but may act as a contributory factor to slight injury or local compression. Arsenic, and occasionally mercury and silver, have been known to produce musculo-spiral paralysis. Diphtheria and influenza are well-known toxic causes of neuritis, and the other infectious fevers act occasionally. Diabetes and enteric fever have been accompanied by paralysis of the circumflex nerve. Paralysis of the musculo-spiral has been observed in enteric fever, articular rheumatism and pregnancy; and rheumatism and gout are believed to operate similarly.

In the *treatment* of injury or compression of a mixed nerve the first thing is to ascertain and remove the cause. Rest, as just mentioned, is of paramount importance. Weak galvanism (6 to 8 m.a.) regularly applied, especially in the form of a limb bath, is of the greatest value, and later this may be combined with faradism and massage. If at the end of many months' perseverance the R. D. is still present, surgery may be summoned to our aid. Surgical treatment followed by the above will sometimes completely restore muscles that have been paralysed and with the R. D. for many years. The distal end of an injured or diseased nerve has been successfully spliced on to another healthy nerve (Purves Stewart and Ballance, Wilfred Harris,[1] Low and others). The treatment of single nerve lesions is also referred to under peripheral neuritis (§ 556) and neuralgia (§ 598).

The SYMPTOMS and CAUSES of PARALYSIS OF INDIVIDUAL NERVES will now be considered. For sensory symptoms see Figs. 162 to 165.

The *musculo-spiral* nerve is paralysed more frequently than any other nerve of the extremities, owing perhaps to its peculiar course and superficial position, and it may be described in detail as a type of localised nerve paralysis. It may be involved in paralysis of the brachial plexus (*vide infra*). The *symptoms* are wrist-drop, due to paralysis of the extensors of the wrist and of the fingers, together with the supinator longus and brevis. Sensation is affected only in a few severe cases. The commonest *cause* is pressure or injury of some kind (group α, p. 1012). Lead poisoning is also a very common cause of paralysis limited to the musculo-spiral nerve; the supinator longus, however, being generally exempt.

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[1] Report of the Clin. Soc. Lond., *The Lancet*, October, 1904.

The OTHER NERVES are less frequently involved singly. The symptoms are given in a table below; the principal causes are as follows:

Circumflex.—Injury to shoulder, pressure, toxic (diabetes, lead).

Long thoracic.—Over-exertion, lifting heavy weights, injury, etc.

Median.—Traumatism, occupation neuroses (dentists, joiners, cigar-makers, etc).

Ulnar.—Pressure and trauma, acute infectious fevers, syphilis, occupation neuroses. Injury or disease of the lowest part of the cervical enlargement may involve only the fibres going to the ulnar nerve; this should be remembered before diagnosing a case as ulnar paralysis. Numbness in this area occurs in tabes dorsalis.

Anterior crural.—Pressure by pelvic or vertebral tumours; toxic (gout, alcohol, diabetes); femoral aneurysm.

Great Sciatic.—Pelvic tumours and inflammation, injury to femur, compression during parturition, toxic (gonorrhœa, gout, rheumatism, fevers, nephritis). Pressure within pelvis often involves only peroneal fibres. An habitually loaded rectum may cause paresis of the muscles and pain in the left leg; but it should be remembered that when sciatica is attended with atrophy, cancer of the sigmoid flexure or rectum or some other pelvic tumour should be considered.

Phrenic nerve.—Diphtheria; injury or disease of the 3rd and 4th cervical roots. The symptoms of paralysis of the phrenic nerve are: (1) dyspnœa on exertion; (2) during deep inspiration the abdomen does not protrude, owing to paralysis of the diaphragm.

TABLE OF ACTIONS OF MUSCLES AND THEIR NERVE SUPPLY.

Nerve.	Muscles supplied.	Defective movement.	Deformity produced.
Posterior thoracic or external respiratory nerve of Bell.	Serratus magnus.	Arm cannot be raised above a horizontal position.	Scapula higher than normal. On attempting to stretch arm forward scapula lifts itself with its inner border "wing-like" from the thorax.
Supra-scapular.	Supra- and Infra-spinatus.	Disturbance in function not pronounced, paralysis of infra-spinatus produces difficulty in writing; of supra-spinatus, fatigue in lifting arm.	Scapular spine prominent when infra-spinatus atrophied.
External anterior thoracic.	Pectoralis major (upper part).	No movement entirely prevented but adduction of arm imperfectly performed.	
Internal anterior thoracic.	Pectoralis major and Pectoralis minor.		
Musculo-cutaneous.	Coraco-brachialis. Biceps. Brachialis anticus.	Forearm flexed with difficulty, especially in supinated position.	Characteristic depression on outer surface of upper arm between insertion of deltoid and origin of supinator longus.
Subscapular.	Subscapularis. Teres major. Latissimus dorsi.	In paralysis of latissimus dorsi forcible backward depression of raised arm is lost. In paralysis of teres major elevation of shoulder with the arm against side is lost.	

Table of Actions of Muscles and their Nerve Supply—*continued.*

Nerve.	Muscles supplied.	Defective movement.	Deformity produced.
Circumflex.	Deltoid. Teres minor.	Arm cannot be abducted nor elevated backward or forward.	Change in the shape of the shoulder; relaxation of shoulder joint develops later.
Musculo-spiral.	Triceps. Anconeus. Supinator longus. Extensor carpi radialis longior. Brachialis anticus.	Elbow, wrist and basal phalanges of fingers cannot be extended; grip weakened; impaired flexion of forearm if supinator longus is involved.	"**Wrist-drop,**" fingers flexed in metacarpo-phalangeal joints; thumb opposed to fingers and somewhat depressed downwards.
Posterior interosseous branch.	All the supinators and extensors of carpus and fingers except supinator longus and extensor carpi radialis longior.		Supinator longus paralysis detected by placing forearm midway between pronation and supination, when flexion against resistance does not bring muscle belly into view.
Median.	Pronator radii teres. Palmaris longus. Flexor carpi radialis. Flexor sublimis digitorum. Flexor longus pollicis. Opponens pollicis. Abductor pollicis. 1st and 2nd lumbricales. Part of flexor brevis pollicis and flexor profundus digitorum.	Flexion of hand with slight force and with ulnar deviation. Fingers cannot be properly flexed at 1st phalangeal joint, while flexion of terminal phalanges only practicable in last three fingers. Pronation of arm lost. Opposition and flexion of terminal phalanx of thumb lost.	Position of hand not markedly altered, generally turned towards ulna and held slightly supinated. Wasting of thenar muscles conspicuous.
Ulnar.	Flexor carpi ulnaris. Adductor pollicis. Muscles of ball of little finger. Interossei. Last two lumbricales. Part of flexor brevis pollicis and flexor profundus digitorum.	Patient can flex hand, but only with adduction towards radius. Inability to flex terminal phalanges of last three fingers and to adduct thumb. Basal phalanges cannot be satisfactorily flexed nor middle and distal phalanges extended. Abduction and adduction of fingers impossible.	"**Claw hand**" most pronounced in 4th and 5th fingers. First phalanx in extreme extension and 2nd and 3rd held firmly flexed; atrophy of hypothenar eminence and of interossei.
Intercostals.	Intercostals. Rectus abdominis. External oblique. Internal oblique. Transversalis.	In paralysis of abdominal muscles forced expiration interfered with; only possible to rise from a reclining position by using arm as a support.	Lordosis; pelvis strongly bent forward; abdomen and nates show up prominently.
Lumbar.	Erector spinæ. Quadratus lumborum.	The back in walking and standing thrown backward; on sitting spinal column is arched convexly backward.	Slight lordosis which disappears on reclining; pelvis raised.

Table of Actions of Muscles and their Nerve Supply—*continued.*

Nerve.	Muscles supplied.	Defective movement.	Deformity produced.
Anterior crural.	Sartorius. Pectineus. Quadratus femoris.	Inability to extend lower leg. Absence of knee reflex. Paralysis of ilio-psoas evidenced by inability to flex hip.	Gait disturbed. Patient stepping carefully, avoiding flexion of knee.
Obturator.	Gracilis. Obturator externus. Adductor longus. Adductor brevis. Adductor magnus.	Adduction and to a slight extent external and internal rotation impaired.	
Inferior gluteal.	Gluteus maximus.	Abduction and particularly extension at hip joint hampered.	In walking leg swings too far inwards, also excessive lifting and sinking of pelvis—waddling gait.
Superior gluteal.	Gluteus medius. Gluteus minimus. Tensor vaginæ femoris.	Loss of abduction and circumduction of thigh.	
Internal popliteal.	Gastrocnemius. Soleus. Tibialis posticus. Flexor communis digitorum. Flexor longus hallucis.	Loss of plantar flexion of foot and toes. Patient unable to lift himself upon tips of his toes. Walking difficult.	Claw position of toes (pied en griffe) pes calcaneus or valgus.
External popliteal.	Tibialis anticus. Extensor proprius hallucis. Extensor longus digitorum. Peronei. Extensor brevis digitorum.	Foot falls from its own weight and cannot be raised, nor can 1st phalanx be extended. Walking difficult, toes scrape the floor.	"**Foot-drop.**" Foot remains in equino-varus position.

§ **565.** II. **Plexus paralysis.** paralysis due to lesion involving a nerve plexus is known mainly by the extent of the paralysis. It is not possible to distinguish nerve root from nerve trunk involvement. There are three forms of brachial plexus paralysis.

(1) *Upper brachial plexus paralysis* of the shoulder and arm (Duchenne-Erb) involves the muscles of the upper arm, namely, deltoid, biceps, brachialis anticus and supinators, sometimes infra-spinatus and subscapularis. It results from tumours or lesions involving the 5th and 6th cervical roots, or by injuries pressing the clavicle against the first rib or spinal column.[1]

(2) *Lower brachial plexus paralysis* of the forearm is much rarer. It involves the muscles of the hand and the flexors of the forearm, sensation being impaired in the region of the ulnar nerve, the inner surface of forearm and upper arm.

(3) *Total brachial plexus paralysis* is very rare and always traumatic.

Brachial neuritis, a toxic ill-defined affection of several of the nerves of the arm, or of the whole of the brachial plexus, is described under pain (§ 598), which is its leading symptom.

Obstetrical paralysis, due to injury during parturition, very often takes the form of upper brachial plexus paralysis; sometimes the facial nerve is injured by the forceps. Recovery generally takes place in course of time. Other forms of birth palsies are referred to under hemiplegia. Lumbar and sacral plexus paralysis is much rarer than brachial, and is due to tumours or disease adjacent to the lumbar or sacral vertebræ. The anterior crural nerve (see table) is generally involved.

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[1] It was this paralysis that Harris and Low successfully treated by cross union of the nerve roots. *Brit. Med. Journ.*, Oct. 24, 1903.

III. **Occupation neuroses**, such as writer's cramp, telegraphist's palsy, etc., may be attended by paresis, but they more often consist of a muscle spasm, under which they are described (§ 578).

IV. **Arthritic amyotrophy** is described under amyotrophy (§ 597). It is due to wasting of some of the extensor muscles following contusion or disease of the joint, sometimes of quite a trivial kind.

§ **566.** b. SPINAL MONOPLEGIAS. Affections of the spinal cord may give rise to monoplegia when the lesion involves the anterior horns or anterior roots. We are therefore in presence of a *lower motor neuron paralysis*, which differs from the last group mainly in the circumstances under which the paralysis occurs, and in some cases the larger number of muscles involved.

I. **Acute Anterior Poliomyelitis** (infantile paralysis) is described under muscular atrophy (§ 595). It is the commonest cause of monoplegia in childhood, and though it may affect more than one limb at the outset, 55 per cent. of all cases settle down into a crural monoplegia. In 23 per cent. of the cases one arm is affected.

II. **Chronic Anterior Poliomyelitis** (progressive muscular atrophy) is met with chiefly in adults as a slow progressive amyotrophy starting at the ends of the limbs, generally in the hands, sometimes in one hand.

III. **Spinal tumours,** or **Pott's disease**, especially when pressing on the nerve roots, in the region of the brachial or lumbar plexus, may commence with monoplegia, and are recognised by—lancinating pains shooting from the back down the limb, the gradual advent and prolonged course of the paralysis, and the concomitant symptoms due to pressure upon the cord (§ 550).

IV. **Syringomyelia** frequently starts as an atrophic paralysis of one, sometimes of both arms, associated with various sensory and trophic symptoms (§ 601).

V. **Amyotrophic lateral sclerosis** and **cervical pachymeningitis** commence as an atrophic paralysis of one or both hands. They are relatively rare diseases.

§ **567.** c. CEREBRAL MONOPLEGIA. Cerebral lesions generally produce hemiplegia: only very rarely a monoplegia. The clinical features here are those of an upper motor neuron lesion, which would at once distinguish it from the two preceding groups.

I. **Focal cortical lesions** producing monoplegias are comparatively rare. Embolism or thrombosis of the anterior cerebral artery (not a common position) produces a crural monoplegia. Occasionally vascular lesions in the aged involve the arm and face centres only. Tumours limited to one of these centres are practically unknown.[1]

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[1] One case has been recorded by Prof. J. M. Charcot and one case by Dr. A. Hughes Bennett.

II. **Hysterical monoplegia** (arm or leg) is less rare than organic cerebral monoplegia. Hysterical monoplegia generally dates from an accident or nerve storm or attack of some kind: and it is usually attended by segmental anaesthesia, *i.e.*, the area of loss of sensation is bounded by a circular line drawn round the limb usually at a joint.[1]

The patient complains of a **widespread paralysis** *or muscular weakness* the case (general debility, p. 766, being excluded) is one of GENERALISED PARALYSIS.

§ 568. **Generalised paralysis** is met with towards the end of quite a number of nervous diseases, but only a few begin with an involvement of all the limbs. We must be careful to exclude the causes of general debility (Chapter XVI.). General paralysis accompanied by stiffness is described under spasm, § 576.

In seeking to DIAGNOSE THE CAUSE of a case of generalised paralysis of this kind one naturally turns *first* to some possible *toxæmic* or *functional* condition such as alcoholism and hysteria.

Secondly, the possibility of some *intracranial disease* such as a basal cerebral or cerebellar tumour, or General Paralysis of the Insane, would next engage our attention, and we should seek for the associated symptoms referable to the cranial nerves or to the mind.

Thirdly, we might suspect some *spinal* or *bulbo-spinal* disease, when the peripheral symptoms, or symptoms referable to the cranial nerves, would be more prominent than the cerebral or mental symptoms.

Toxic or functional.

1. Alcoholism.
2. Diphtheria, syphilis, hydrophobia.
3. Hysteria.
4. Myasthenia gravis.

Intracranial lesions.

1. General Paralysis of the Insane.
2. Diffuse basal tumours.
3. Cerebellar tumours.
4. Cerebral pachymeningitis and pachymeningitis hæmorrhagica.
5. Infantile diplegia cerebralis.
6. Encephalitis.

Spinal and bulbo-spinal lesions.

1. Disease or injury high up in the spinal cord.
2. Cervical pachymeningitis.
3. Disseminated sclerosis and other chronic degenerative conditions.
4. Bulbar paralysis.
5. Landry's paralysis.
6. Diffuse myelitis.
7. Acute anterior poliomyelitis.

Idiopathic muscular atrophy.

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[1] A very typical case forms the subject of a clinical lecture by the author in the *Clinical Journal*, May, 1904

Among the TOXIC and FUNCTIONAL causes, (1) **Chronic alcoholism** is not infrequently followed by a generalised weakness passing on to paraplegia. It is probably due to a generalised toxic degeneration of the nervous system, or to peripheral neuritis.

(2) Other causes of peripheral neuritis such as **diphtheria**[1] may also produce general paralysis. The **syphilitic** toxin, however, produces General Paralysis of the Insane. In the last stages of **hydrophobia** general paralysis ensues.

(3) In **hysteria** I have occasionally met with a flaccid paralysis coming on suddenly and affecting all the limbs.

§ 569. (4) **Myasthenia gravis** (Asthenic Bulbar paralysis) is a rare condition probably toxic in origin, consisting of progressive weakness of all the cerebro-spinal muscles. It was first described by Willis in "The London Practice of Physic" in 1685, and was studied again by Sir Samuel Wilks in 1877, and Erb in 1878. About 60 cases had been recorded up to 1900.[2]

Symptoms. 1. The patient easily becomes tired and the muscular weakness is always worse in the evening. All the voluntary muscles of the body are affected, but especially those of the face, eyes, and neck. Bilateral ptosis is present in about 80 per cent. of the cases, and all the ocular muscles are weak (ophthalmoplegia externa). The face is expressionless, and there is often a difficulty of swallowing and of articulation. A nasal speech after talking awhile, and a difficulty of mastication after starting to eat are other examples of the rapid fatigue of the muscles. Paroxysmal dyspnœa may supervene. All the symptoms vary in intensity from time to time, but the patient gets gradually weaker. 2. The reaction to faradism is characteristic, for the muscles become exhausted in a very short time; although the muscles will contract at the moment when the terminal is first applied they soon become completely flaccid again, and after several applications may fail altogether to respond. This, the myasthenic reaction, is quite peculiar to the disease. 3. There is no fibrillation, no obvious muscular atrophy (till late in the disease), no reaction of degeneration and no sensory changes.

Diagnosis. Cases may be mistaken for bulbar paralysis, in which, however, the ocular symptoms are wanting. Diphtheritic paralysis is like myasthenia, but the myasthenic reaction is wanting; so also in neurasthenia, hysteria, and the early phases of chronic degenerative lesions. In Addison's disease there are pigmentation, emaciation and other symptoms.

The *prognosis* is grave, the disease is insidious, and may last for several years, but is mostly in the end fatal, either from sudden asphyxia, or some intercurrent affection.

The *etiology* is very uncertain. Young persons are mostly affected. The generalised character of the symptoms and the resemblance of myasthenia to diphtheritic paralysis suggest a toxic origin, and it has been suggested that the toxin, whatever it is, specially affects the motor nerve endings in the muscles.

For the *treatment*, rest, massage, full doses of strychnine, iodide of potassium and mercury may be tried.

INTRACRANIAL LESIONS mostly produce hemiplegia, but there are 3 conditions which may produce generalised paralysis.

1. **General paralysis of the insane,** which is a chronic degenerative change in the cerebral cells, gradually passes on to a generalised paralysis.

2. **Diffuse basal tumours** involving the pons or the peduncles may cause generalised paralysis, but in that case the cranial nerves are also involved (§ 602 *et seq.*).

3. **Cerebellar tumours** may produce general paralysis, associated with nystagmus and a characteristic gait (§ 571).

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[1] Dr. F. E. Batten (*Brit. Med. Journ.*, Nov. 19, 1898) has shown that very few of the nerves really escape in diphtheritic paralysis.

[2] Dr. Harry Campbell and Dr. Edwin Bramwell, *Brain*, 1901.

4. **Cerebral pachymeningitis** produces a vague generalised paresis and mental weakness.

5. **Hæmorrhage** or **injury** at birth may cause paralysis of both arms and legs (Infantile spastic diplegia).

6. **Encephalitis** (polio-encephalitis) is a morbid condition to which attention has lately been directed by Dr. F. E. Batten and others (Dr. F. E. Batten—"Acute Polio-myelitis and Encephalitis," *The Lancet*, Dec. 20, 1902, and "Polio-encephalitis Inferior," Trans. Path. Soc., London, vol. 54, part iii., 1903), in which miliary foci of thrombosis affect the cortical cells in the same way as anterior polio-myelitis affects the anterior horns. The subjects are mostly children. The symptoms necessarily differ with the position of the mischief, but a generalised weakness is not uncommon, and this may be associated with symptoms of insular sclerosis, or ataxy.

It is doubtful whether the above should be called encephalitis. But I have occasionally seen what appears to be a diffuse inflammation of the brain substance. I remember a case of recent syphilis in a man of about 31, who was admitted into the Infirmary with symptoms of cerebral irritation—restlessness, muttering delirium, general weakness, commencing optic neuritis, who died a few days later comatose. After death the organs of the body and the meninges were healthy, excepting for congestion, but the brain was deeply injected, and its substance extremely soft, so that it could be washed away under the tap, and was of a brownish tint. Under the microscope the cortical cells were swollen, blurred, and granular. The cerebral arteries appeared to be quite healthy.

Among SPINAL and BULBO-SPINAL LESIONS (1) **Pott's disease** and **spinal tumours** high up in the cord may involve all four limbs, the mind remaining clear. Injury at birth affecting the upper part of the cord causes **infantile spastic paralysis** (§ 555) which is more usually a paraplegia only.

2. **Cervical pachymeningitis,** amyotrophic lateral sclerosis, and progressive muscular atrophy may produce a like effect.

3. Disseminated sclerosis, tabes and other **degenerative spinal lesions** may terminate in generalised paralysis.

4. **Bulbar paralysis** (glosso-labio-laryngeal palsy) may affect all the limbs when it comes on acutely. Chronic cases are only affected by generalised paralysis in their final stages.

§ 570. 5. **Landry's Paralysis** is a rare condition of ascending flaccid paralysis of which the pathological explanation is obscure.

The *symptoms* start with flaccid paralysis of the legs rapidly extending to the trunk, arms and neck, and cranial nerves in some cases. It usually terminates fatally from involvement of the respiratory muscles, in two days to two weeks. There are no (or very slight) sensory changes, no muscular atrophy, no trophic or electrical changes, and no loss of sphincter control. The spleen has been enlarged in some cases.

Etiology. It occurs chiefly in males between 20 and 30. It bears considerable resemblance to a rapid peripheral neuritis, and it has been suggested that it is due to an acute toxæmia of the lower motor neuron. A tetracoccus has been found in the spinal fluid by lumbar puncture (Farquhar Buzzard).

6. **Acute diffuse myelitis** may rapidly ascend the spinal cord, and cause an acute generalised paralysis. There is anaesthesia and loss of sphincter control, but no muscular atrophy (§ 552).

7. Acute spinal paralysis of adults is really an **acute anterior poliomyelitis** similar to that affecting children (§ 595). Wasting is rapid, with R. D., and recovery is never complete. A sub-acute form is also described.

General paralysis associated with extreme wasting of the muscles is seen in the later stages of the **idiopathic muscular atrophies,** and the peroneal type of myopathy (§ 597). The gradual onset in youth, the progressive course of the disease, and the hereditary history aid the diagnosis.

GROUP IV. β. INCO-ORDINATION AND DISORDERED GAIT.

§ 571. The **gait** of all patients suffering from motor defect should, if possible, be studied by the physician: it will often teach us a great deal about the malady. The defect may be due to muscular weakness, to muscular rigidity, to clonic spasm, to true inco-ordination, or to want of balancing power. The most

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characteristic disorder of the walk occurs in the *inco-ordination* of tabes dorsalis and the sinuous or *reeling gait* due to want of balancing power in cerebellar lesions. The anatomical remarks on this subject (§ 502) may help to explain what follows.

The various diseases in which disordered gait is met with may be grouped as follows :—

a. INCO-ORDINATION (or ataxy) is met with most characteristically in —(1) *locomotor ataxy* (tabes dorsalis), in which there is an exaggerated movement, a stamping gait, with the feet wide apart, lifted high and brought down forcibly, *i.e.*, the patient being unable to co-ordinate his movements. It is met with in other diseases affecting the posterior columns, namely, (2) *spinal tumour* affecting the posterior columns, especially in the lumbar region : (3) *chronic posterior spinal pachymeningitis ;* (4) *Friedreich's hereditary ataxy* (rare) ; (5) *ataxic paraplegia* (rare) ; and (6) other conditions where the *posterior columns* are involved primarily or secondarily.

b. REELING GAIT or swaying like a drunken man, is met with most typically in (7) *cerebellar disease.* It also occurs in patients who are the subjects of (8) frequent or continuous *vertigo* either from circulatory or other causes.

c. The SPASTIC GAIT is a stiff gait owing to the stiffness and rigidity of the legs, due to lateral sclerosis. By degrees the patient takes to walking on tiptoe, the toes turn in, and are scraped along the ground. Later still we get what is called cross-legged progression, owing to the predominance in the contracture in the adductor muscles. This may occur on one or both sides. On *both sides*, it is met with in the various (9) rigid or *spastic paraplegias* (§ 555), and in ataxic paraplegia. On *one side* it occurs in (10) organic *hemiplegia* owing to descending sclerosis, in which the spastic character of the gait induces a circumduction of the rigid limb as it is brought forward in walking, which, as Sir Benjamin Brodie pointed out many years ago, is in strong contrast with the flail-like flaccidity of many functional hemiplegias. It must be remembered, however, that this distinction only applies to hysterical hemiplegia when the paralysis is flaccid ; some cases are attended with rigidity, and in these the distinction does not obtain.

d. The FESTINATION GAIT, in which the patient is hurried forwards, as he walks and shuffles along the floor, his face looking fixedly forwards, is met with in (11) *paralysis agitans,* and to some extent in old age.

e. The HIGH-STEPPING GAIT is met with typically in well-marked (12) *peripheral neuritis* and other amyotrophic (lower neuron) paralyses attended by foot-drop, on account of the predominance of the paralysis in the extensor muscles, the patient, so to speak, flinging the foot upwards instead of raising it. It is also met with in (13) *pseudo-hypertrophic paralysis.*

f. There is a peculiar "JAUNTY" walk in (14) *chorea,* associated with excessive arm movement, which is very characteristic. Other tremors (§ 583) may render the walk peculiar.

g. There is a very characteristic ATTITUDE and gait, with the head and arms hanging forward owing to the weakness of the neck muscles, in (15) *post-diphtheritic paralysis* of childhood.

h. The WADDLING gait is met with in (16) *congenital hip dislocation*, advanced *rickets*, *achondroplasia*, all conditions of *dwarfism*, and in *coxa vara*. This last named is a peculiar congenital condition, in which the neck of the femur forms a less obtuse angle than is usual, and causes the patient to sway from side to side as he walks.

i. The LIMPING GAIT is met with as a result of (17) *infantile paralysis*, and in any (18) injury or *joint affection* confined to one side.

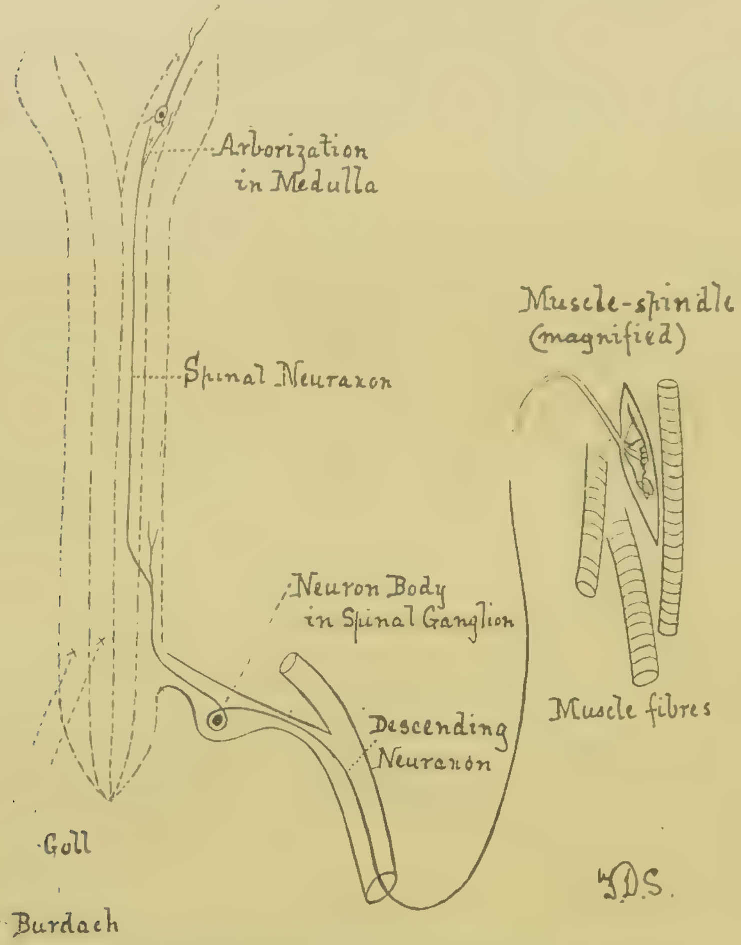

Fig. 166.—Diagram of the spinal cord and muscle-sense neuron to show the PATHOLOGY OF TABES DORSALIS. The sites of initial degeneration are at the ends of the ascending (spinal) neuraxon (viz., its arborization in the medulla) and the descending neuraxon (viz., its termination in the muscle-spindle).

§ 572. I. **Tabes dorsalis** (locomotor ataxy) may be defined clinically as a very chronic disease commencing with disturbances of the muscle sense and various other derangements of sensation and of the cranial nerves, and terminating in total abolition of the faculty of co-ordinating the voluntary movements; muscular power usually remaining intact until near the end.

Anatomically, until recently tabes dorsalis was described as a primary sclerosis of Burdach's columns chiefly in the lumbar region, combined with a similar process involving more or less of the posterior spinal roots, the spinal ganglia, and sometimes the posterior horns, and giving rise to an ascending sclerosis in Goll's columns, the antero-lateral tracts, and occasionally the cerebellar tracts above. Other parts are also sometimes involved; thus, Charcot mentions that the cells in the anterior horns are affected in cases accompanied by joint disease. This, ever since the days of Todd, was regarded as the essential lesion of the disease, but, unfortunately for this view, there have been at different times cases recorded[1] in which, although the patient revealed all the most typical symptoms of the disease during life, there were no such lesions found after death. During the past few years valuable contributions have been made to our knowledge concerning the structures which are subservient to muscular sense, and about the real lesion in tabes. A little fusiform structure called the muscle spindle has been known to physiologists for half a century. Measuring about three by one-third of a millimetre, these spindles were found amongst the muscle fibres in all the muscles of the body, excepting the diaphragm, the intrinsic muscles of the tongue, and (?) those of the eyes. Each consists of a laminated sheath of white fibrous tissue, containing a lymph space, in which lie striated fibres resembling muscle fibres, only one-third of their diameter. Nerve fibres in great profusion enter the spindle, and Professor W. S. Sherrington[2] has shown not only that these nerve fibres are connected with the posterior roots, but that they convey afferent impulses; and there is no doubt, as Kerschner and others maintained, that these are really the end organs of the muscle sense nerves. Dr. F. E. Batten[3] has shown that these muscle spindles are diseased in tabes, the intra-fusal nerve endings and contiguous muscle fibres undergoing degeneration, and that the same changes are absent in primitive myopathies, progressive muscular atrophy, and infantile paralysis. Now whenever the nutrition of a neuron is impaired, the peripheral endings of its processes are the first to undergo degeneration. Applying this principle to the pathology of tabes in the light of Sherrington's and Batten's researches, the condition of affairs may be depicted in Fig. 166. The cell body of the muscle-sense neuron is found in the posterior ganglion, and it has an upper or spinal neuraxon, and a lower or peripheral neuraxon; and in tabes we now know that the degeneration starts at the peripheral end of the latter. We have good reason to believe, though the grounds are not quite so sure, that the same degenerative process attacks the termination of the upper neuraxon, which is in association with the arborizations of the cerebro-bulbar neurons. In view of these valuable researches we can now understand why, in some of the cases of tabes, no sclerosis of the spinal cord is found, by supposing that such cases died before the degeneration had spread to this position. It also helps to explain why in this essentially spinal affection, which starts with ataxy of the limbs (owing to degeneration of the lower neuraxon), we are liable to have so many cranial manifestations (owing to degeneration of the upper neuraxon). And, finally, it also helps us to explain why, in the long pre-ataxic stage, the chief manifestations are associated with sensory disturbances, because we may fairly assume that the other sense neurons of the lower level are similarly affected by the slow degenerative process. The presence of sclerosis in the spinal cord in such cases is explicable on the modern pathological view of interstitial fibrosis, which is secondary to the nerve degeneration.

The *symptoms* of tabes run a prolonged course of 10, 20, or even 30 years, and they may be divided into three stages—pre-ataxic, ataxic, and paralytic.[4] It is of the greatest importance to recognise the malady in its earliest phase, because at that time treatment is more efficacious, and the disease may in some cases be arrested. The symptoms are most varied; there is really only one that is always present—loss of knee-jerks.

(a) In the *pre-ataxic stage* the most important and characteristic symptoms consist of "lightning pains"; vague disturbances of sensation; changes in the pupil, and other ocular symptoms. Any of these, combined with loss of knee-jerk, is almost sufficient for a diagnosis. (1) The "lightning pains," or crises, of tabes are characterised by being erratic, evanescent, recurrent, and sometimes periodic. Tabes should be suspected in any neuralgias having these characters, especially in the sciatic nerve, but any sensory nerve may be affected. A "girdle pain" round the waist is another frequent symptom in the early stages—6 years before any other symptom in a case I saw recently. Pains also occur in the situations of the various viscera, and come on in attacks, which the French writers have named "crises." Thus, there may be attacks of gastric pain, followed perhaps by vomiting (gastric crisis); or rectal pain, which may be attended by constipation (rectal crisis); and so on. Frequently the bladder is the organ affected, sometimes with pain (vesical crisis), more often with a hesitation in passing water, or with actual retention, and the patient or his doctor may think that there is a stricture or enlarged prostate. Thus cases of tabes may frequently come before the surgeon. Laryngeal crises are occasionally observed, and consist of spasmodic attacks of laryngeal dyspnœa, often coming on at night. These may be mistaken for asthmatic attacks, and are very alarming, but the patient recovers in an hour or so. All these pains are alike in being erratic, evanescent, and recurrent. (2) Numerous and remarkable are the many different anæsthetic and paræsthetic symptoms observed in tabes. One of the commonest consists of scattered patches of anaesthesia, especially along the ulnar borders of the hands, and on the soles of the feet, giving rise to a sensation of "treading on cotton wool." Sometimes there is analgesia without anæsthesia of the whole or part of the body. Sometimes there is thermal anæsthesia; sometimes delayed sensation; sometimes misplaced sensation (allocheiria): but the strangest of all, perhaps, is what Duchenne described as the "masque tabetique," in which the patient has anæsthesia of the nose, lips, or face, which gives him the impression that he has no face. Perversions or abolitions of taste or smell are not uncommon, and diminution or increase of the sexual power (3) The ocular symptoms are very important, and the most frequent of these is transient diplopia, due apparently to a transient weakness of one or other of the ocular muscles. My experience bears out the statement that quite half the cases of tabes at one time or another suffer from diplopia. The pupils present very characteristic signs. The most typical of these is the Argyll-Robertson pupil, *i.e.*, loss of mobility to light, but not to accommodation.[1] Another very common change is inequality of the pupils; or they may both be very contracted, or both immobile to all forms of stimulus. Another frequent manifestation is primary optic atrophy, coming on with a greyish appearance of the optic disc, and terminating in white discs with clearly-defined margins. Any of these symptoms may precede the ataxy by 10 or more years. In my experience, if optic atrophy is an early symptom, the ataxy is very late in developing; in one of my cases it never developed at all, though all the other symptoms were present. (4) The absence of knee-jerk is certainly among the earliest symptoms; it is the only constant and invariable symptom of the malady. (5) Various trophic disturbances are now known to be associated with tabes, thanks chiefly to the observations of Charcot. It was he who pointed out that a particular kind of osteo-arthritis, now called "Charcot's joint disease," may be present in this malady, and it may occur quite early, at a time when other symptoms are in abeyance. The joint becomes swollen, but usually it is pale, and free from pain. Indeed, there is such a complete absence of pain, heat, and redness, the three classical symptoms of inflammation, that the joint, the hip for instance, may become quite disorganised or dislocated ere the patient pays much heed to it.[2] In the knee-joint, which is the favourite situation, the swelling is less likely to be overlooked. The occurrence of such an arthritis, combined with the pupillary changes and absent knee-jerk, are alone enough to establish the diagnosis. The nails may be affected with a sort of painless ulceration; the teeth may be similarly affected and drop out, or perforating ulcers of the foot may occur. All these lesions have the same slow chronic characters. It will be observed that the pre-ataxic symptoms fall into two categories—

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[1] *e.g.*, Dr. A. Hughes Bennett, Clin. Soc. Trans., vol. xviii.; Déjérine, and others.

[2] *Journal of Physiology*, 1896-7.

[3] *Brain*, Autumn number, 1898.

[4] It is interesting to notice that in the course of years our knowledge of tabes has, as it were, gradually shifted backwards, so that what were formerly regarded as the essential symptoms of the disease are now looked upon as complications or sequelæ. At first the disease was classed as a paraplegia; now we know that paralysis of the legs only occurs at the termination, and not always then. Next, it was regarded as an ataxy. Finally, we are coming to regard even this ataxy as an advanced symptom, and at the present day we concentrate our attention on what is called the "pre-ataxic stage," which consists of muscle-sense and other sensitive and sensorial changes.

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[1] The student can recollect this by the two A's—Act to Accommodation, not to light.

[2] In an interesting case narrated by Charcot the patient was doing his military service, and casually found that he could not march as well as the others, and on examination it was ascertained that the two hips had gone on to dislocation. "Nouvelle Iconographie," tome v.

central or cranial, and peripheral, and this is now explained by the researches of Sherrington and Batten (Fig. 166). As to their relative value, if the patient be a male, of an age between 30 and 50, and present symptoms taken from any two of the above groups, I believe one is justified in diagnosing the disease.

(b) *The ataxic stage* consists of the above symptoms, which gradually become emphasized in the course of years, combined with (1) the characteristic walk described above, and (2) loss of equilibrium on standing. The normal power of the muscles remains, and their nutrition is often remarkably good, but the patient cannot control them. The typical ataxic gait has been described, and is easily recognisable when once seen (§ 571). These patients find a difficulty in starting to walk, but once started, they may improve as they go on, up to a certain point. They also have great difficulty in turning round, and the late Dr. Hilton Fagge mentioned an amusing incident of a patient who once explained his unpunctual arrival at the hospital by his having started in the wrong direction, and being compelled to continue until he met someone who could help him to turn round and start the other way. The arms are much less affected than the legs, at any rate until quite late; the patient can write or pick up a pin, but there is often a difficulty in touching the nose when the eyes are closed. The loss of equilibrium on attempting to stand with the eyes shut and the heels together is known as Romberg's sign, and is very characteristic of this disease. The explanation of this sign is given in § 502.

(c) The *terminal paralytic stage* is really the stage of *complications*, and it may be many years before this stage sets in. The mind remains clear in most cases until quite the end, and the patient may be able to conduct his business for 10, 20 years or more after his locomotion has become imperfect. By degrees the inco-ordination becomes extreme, and by-and-bye paralysis supervenes, generally, though not always of an atrophic form. Bladder complications are frequent, and various other visceral conditions supervene; death results from these or bedsores, not from the disease. Perhaps the most frequent cause of death is pneumonia. Bulbar paralysis may supervene, but one of the most frequent of the nerve complications of tabes is general paralysis of the insane (paralytic dementia); and in visiting an asylum, it is remarkable to learn what a large number of general paralytics have commenced as cases of tabes. Many varieties and transitional forms are met with in these two diseases among out-patients, and give rise to much difficulty unless you remember this frequent association.

Causes. (1) *Age.* Like general paralysis of the insane, tabes is almost entirely confined to adults between the ages of 25 and 40, and (2) it is almost entirely confined to the male sex, certainly over 90 per cent.[1] (3) Syphilis can very generally be traced in the history of cases of tabes, and in spite of the fact that iodides and mercury fail so completely in the treatment of tabes, there is very little doubt that tabes is really a slow parasyphilitic degeneration of the muscle-sense neurons. (4) A history of neuropathic antecedents, and especially alcoholism in the parents is often present. Prof. J. M. Charcot[2] insisted very much on the potency of alcoholism in the father as a cause of this and many other nerve affections. Sometimes a family history of other nervous maladies can be traced. (5) Prolonged bodily fatigue, especially if combined with exposure to cold and malnutrition, act both as predisposing and exciting causes. The disease is frequent in postmen, commercial travellers, and others who lead an active life. This is interesting in connection with the theory of origin in forced functioning just referred to. Among the rarer causes, venereal excess, prolonged grief, anxiety or mental strain may be mentioned.

The *diagnosis* of tabes in typical cases—and tabes is one of the few diseases of the nervous system which usually conforms to a type—is not difficult excepting in the very earliest (pre-ataxic) stage. The age, sex, and syphilitic history are very characteristic, and the only disease apt to be confused with it at this time is general paralysis of the insane, which however usually presents the mental peculiarities and muscular tremor that are wanting in tabes. The occurrence of compound cases has been mentioned. In the ataxic stage it may have to be differentiated from lumbar tumour, pachymeningitis, ataxic paraplegia and other diseases which will be mentioned below.

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[1] The fact of tabes predominating so markedly in the male sex is interesting in connection with the theory of "forced functioning," which may be briefly stated thus:—Prolonged forced functioning of any nerve structure (especially if combined with malnutrition) results, in course of time, in atrophy and overpowering of this structure by the surrounding tissues, and its consequent degeneration. Males use their co-ordinating and muscle-sense apparatus much more than females.

[2] "Chaque goutte de liqueur séminale d'un alcoolique contient, en germe, la famille neuropathique tout entière." "Nouvelle Iconographie de la Salpetrière."

The *prognosis* of tabes has hitherto been regarded as hopeless, at any rate for cure, though the disease occasionally lasts throughout a patient's lifetime without materially shortening it. But by degrees, by careful attention to clinical detail, we have learned to recognise it in its earlier stages, and much may certainly be done at that time to arrest or delay the degenerative process. The prognosis rests chiefly on three points; first upon the rapidity of development of the symptoms; secondly, on the causes in operation, especially when some of them are removable; and thirdly (the most important point) the stage at which the disease is recognised.

Treatment. (a) For the *curative treatment* all anti-syphilitic treatment is almost useless, though mercurial inunctions and injections may be tried in the early stages; iodides are certainly useless. The leading principles which I have found most useful are, the cessation of function of the deranged structures, a liberal dietary, combined with galvanism. Perfect rest in bed should be enjoined, the patient not being allowed even to stand for an instant. The dietary should contain a large proportion of fatty food; tonics may be administered to promote the assimilation especially cod-liver oil, as being, in my opinion, the remedy which is the best "nerve food." Electricity requires great judgment, and should not be applied to cases which are irritable, or rapidly advancing. The best way, I have found, is to apply an ascending current to the spinal cord, the negative pole being fixed on one side of the back of the neck and the positive pole being shifted from place to place down the back of the spine on the other side, resting half to one minute at each place. The strength of the current should be very moderate. Long sittings and strong currents should be avoided, but a sitting should be held every day. The system of exercises elaborated by Dr. Frænkel has for its object the education of the lost power of muscular co-ordination. I have applied this method of treatment in early and suitable cases with marked benefit. Belladonna and

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ergot may be given in cases where congestion of the cord is suspected from the presence of spinal pain and rapidly developing symptoms. Arsenic, silver, and other metals I believe to be injurious; at least I have known patients go back under their use. Didymin and cerebrin are two remedies which are still on their trial. Patients should certainly avoid anything like fatigue of mind or body. Sexual excess, exposure to cold, alcohol and tobacco should be avoided. (b) In regard to *symptomatic treatment* the most important symptoms calling for treatment are the lightning pains. Morphia must be forbidden, for with a recurrent pain like this the habit is bound to be developed. On the other hand, the same treatment which cures or modifies the disease also relieves the pains. Phenacetin, antipyrine, hyoscyamus, belladonna, physostigmine, Indian hemp, and other analgesics, combined with rest and warm baths are often successful.

Whenever the **posterior columns** of the spinal cord are affected, whether primarily or secondarily, an ataxic gait and other symptoms resembling tabes may arise. Severe anæmias and other toxic debilitating conditions may be complicated, as Dr. James Taylor and others have shown, by posterior sclerosis and ataxic gait. In alcoholic subjects there is at first a more or less transient ataxy, which later on, as posterior sclerosis ensues, becomes permanent. There are four named DISEASES OF THE SPINAL CORD, all of which compared with tabes dorsalis are relatively rare, but give rise to inco-ordination of the gait resembling tabes, viz., Spinal Pachymeningitis, Spinal Tumour involving the posterior columns, Friedreich's Disease, and Ataxic Paraplegia.

§ 573. II. **Spinal pachymeningitis**, or chronic thickening of the meninges, is apt sometimes to be confined to, or at any rate to predominate in, the **posterior** region of the theca vertebralis, and in some cases I have seen gave rise to symptoms which it was impossible to distinguish from those of tabes dorsalis. I believe that many cases of so-called tabes cured by iodide were possibly instances of this affection of syphilitic origin. One is assisted in recognising this condition if there is the long history of *continuous* pain and tenderness in the spinal column, and continuous pain shooting down the spinal nerves; and sometimes by the age and sex of the patient.

Posterior spinal pachymeningitis is extremely chronic, and not infrequently the symptoms are few and ill marked; on which account such cases very rarely find their way into hospitals. They get into the infirmaries, however, and my experience at the Paddington Infirmary led me to the conclusion that the disease is fairly common. At a meeting of the Neurological Society of London held at the Paddington Infirmary in 1890, I was able to show the cords of five fatal cases, and three examples of the affection during life. Here is a fairly typical case—a woman, æt. 72, was admitted into the Infirmary in January, 1890, with all the classical symptoms of tabes, preceded and accompanied by very severe lightning pains. The history, briefly, was that for three years prior to admission she had suffered from paroxysmal pain shooting down the legs and various other places, and that she had had other attacks which resembled gastric crises. For two years prior to admission she had suffered from ataxy which became progressively worse, and at last so marked that she was unable to stand. Many careful observers saw the case; all had no doubt that it was an example of locomotor ataxy occurring in a woman. She died of pneumonia in June, 1891, and at the autopsy, though the pia mater and arachnoid on the anterior surface of the cord was normal, that on the posterior surface was thickened, and opaque. This thickening was irregularly distributed from end to end, and was much more marked in certain patches of three inches in length in the mid-dorsal and in the lumbar regions, in which positions it was about the thickness of a piece of washleather, and showed commencing calcareous plates. These patches were firmly adherent to the posterior aspect of the cord, and the posterior columns beneath were sclerosed as in case of advanced tabes.

III. **Spinal tumour** (intra- or extra-rachidian) pressing on the back of the cord, especially in the *lumbar region*, is often attended by ataxic symptoms closely resembling those of tabes. It may be distinguished from tabes, (i.) by the absence of the pre-ataxic symptoms above mentioned; (ii.) by the fact that it is preceded by severe, constant, neuralgic pains in the spine, spreading upwards from the lumbar enlargement; (iii.) as in other cases of spinal tumour, all the symptoms—which, be it noted, include actual weakness and often stiffness of the legs—have a tendency to be unilateral, *i.e.*, predominate in one or other leg (§ 550).

§ **574.** IV. **Friedreich's disease** (Hereditary Ataxia), a rare hereditary condition occurring in children, often sisters and brothers of the same family. These cases have 5 differential characters. (i.) There is inability to stand with the eyes shut, loss of patellar reflexes, and other symptoms characteristic of tabes. The disease, in short, presents a mixture of the symptoms of disseminated sclerosis and tabes; and pathologically it has been shown to be a postero-lateral sclerosis. There are, however, no lightning pains, no crises, and no Argyll-Robertson pupil. (ii.) Tremors of an ataxic nature come on later in the arms, so that the hand in approaching the mouth does not reach its goal, and is accompanied by jerky, irregular movements of the head and neck. (iii.) The speech is impaired in the same manner as in disseminated sclerosis. (iv.) Nystagmus is present in most cases. (v.) There is usually no mental change. The muscles become weakened, and deformities such as scoliosis and talipes ensue.

V. **Ataxic paraplegia** is another rare affection, which corresponds, in my belief, to the spasmodic tabes dorsalis of some authors. It is due to sclerosis affecting the posterior as well as the lateral columns, and it results in symptoms of the two lesions. It is differentiated from tabes by the spastic rigidity, increased knee-jerks, and ankle-clonus. The Argyll-Robertson pupil and lightning pains are absent. As time goes on the rigidity becomes more marked.

§ **575.** VI. **Cerebellar tumour,** in which the gait is sometimes ataxic but more often staggering, is often very obscure, but it may be suspected in presence of a group of four symptoms, viz., a reeling gait, headache, vertigo, and nystagmus, especially when those symptoms occur in childhood. The walk is fairly characteristic, having a reeling character, and the patient tends to sway from side to side like one intoxicated. But sometimes, especially when one lateral lobe is affected, the patient tends to fall over towards the side of the lesion. There is frequently weakness, without actual paralysis, sometimes hemiplegic, but more frequently paraplegic or general in its distribution. The knee-jerk does not help us, for it may be absent, increased, or normal. The cranial nerves most affected are the fifth, sixth and the optic nerve. Symptoms occasionally present are forced movements, of the neck for example, pulling down the head towards the side on which the lesion is situated in tumour of the lateral lobe. Other occasional symptoms are attacks of syncope, palpitation, and dyspnœa, which are of considerable gravity, as they usually indicate pressure on or spread to the medulla, though in one instance of this kind the patient (on whom I made the autopsy) lived for 1 year and 10 months after the onset of these symptoms.

The *prognosis* and *treatment* of cerebellar tumour is that of other intracranial tumours (§ 546). Its deep-seated situation and the proximity of the medulla render it one of the most serious of intracranial tumours. In a few cases the tumour has been successfully removed.

GROUP IV. γ. INCREASED MUSCULAR ACTION.

Involuntarily increased muscular action may be—

Continuous, when it is known as rigidity or *tonic spasm* . § 576

Intermittent, when it is known as *tremor* if the movements are small and vibratile, and *clonic spasm* if the movements are large . . . . § 583

Attacks of violent muscular movements, associated with more or less disturbance of consciousness, are known as *convulsions* . . . . § 591

The patient presents a continuous stiffness or **rigidity** *in the affected muscles*—TONIC SPASM.

§ 576. **Tonic** (continuous) **muscular spasm** or rigidity is indicative of an irritative lesion, functional or organic, in some part of the motor tract, or a morbid irritation in the muscular substance. It occurs in the following conditions—

I. Paralytic rigidity (early and late).
II. Hysterical rigidity.
III. Occupation cramp.
IV. Cramp.
V. Tetanus.
VI. Tetany.
VII. Hydrophobia.
VIII. Thomsen's disease.
IX. Arthritic rigidity.

The 1st of these causes is of organic origin ; the 2nd, 3rd and 4th are functional ; the 5th, 6th and 7th are toxic ; the 8th is due probably to some hereditary defect ; and the 9th is probably reflex.

§ 577. I. **Paralytic rigidities of organic origin** are associated with paralysis and are of three kinds as exemplified in hemiplegia.

1. *Early rigidity* of the muscles is that which comes on with the paralysis in cases of hemiplegia due to hæmorrhage, or within the

next few days in association with irritative lesions such as pressure on the anterior spinal roots. The rigidity of haemorrhage comes on suddenly and passes off gradually in the course of a week or so. The rigidity of other irritative lesions only passes off when the irritation is removed or the nerve tracts destroyed.

2. *Late or spastic rigidity* of the muscles is that which comes on gradually in the course of a month or so in all cases of paralysis due to lesions of the upper motor neuron. It is met with typically in hemiplegia, spastic paraplegia, and all lesions followed by *descending lateral sclerosis* in the spinal cord. It is always associated with increased knee-jerks, ankle-clonus and Babinski's sign; tremors or clonic muscular spasms or athetosis may also be present. It comes on gradually and increases progressively. It diminishes during sleep. Chloral hydrate relieves this kind of rigidity, which is sometimes very troublesome and painful in hemiplegic cases.

3. *Organic contracture* ensues in all paralysed muscles in the course of years, whether of the upper or lower neuron type (such as hemiplegia and infantile paralysis respectively). It is due to an atrophic fibrosis of the substance of the paralysed muscles.

II. **Hysterical rigidity** may easily be confounded with 1 and 2 above, unless the history is accessible. It generally comes on quite suddenly after an emotional storm. It is often of limited extent, involving perhaps only the wrist joint. It may pass off as suddenly as it came. It rarely lasts long, but may occasionally persist for years. This rigidity is said to persist during sleep. The patient, who is a female, presents other signs of hysteria.

§ 578. III. **Occupation Cramp,** and other **Occupation Neuroses.** We have already seen that exhaustion produces muscle-cramp. The continual over-use of a certain group of muscles is apt to produce 5 symptoms, at first localised to that part, but tending ultimately to spread to other muscles, if the cause continue in operation. In order of frequency, they are, (1) tonic spasm, (2) paresis, (3) pain, (4) tremor, and (5) in some cases either hypertrophy or atrophy follows.

Writers' cramp is the most frequent example, and is taken as a type, but what follows will apply almost equally to other occupation neuroses. (i.) *Tonic spasm.* In scriveners' palsy, after writing for some time, the fingers get so stiff that the patient cannot write. Sometimes the spasm is very painful, and occasionally there are twitchings. The character of the writing alters, and in the course of weeks or months the slightest attempt at writing produces a tonic spasm in the muscles used for that purpose. For a long time *tonic spasm appears only on attempting to write*, the parts in the intervals being quite free from symptoms, but in the later stages the spasm may become persistent or start spontaneously. In the later stages also the spasm is apt to spread from the hand to the arm, shoulder, neck, and other parts. In the early stage the general uses of the limb are unimpaired, and a patient affected with writers' cramp may paint, or play the flute, but in most cases other delicate manipulative procedures cannot be performed with the same precision as formerly. (ii.) The *power* of the grasp, as tested by the dynamometer, is said to be normal, but this is not a delicate test of the small muscles in writing. By careful examination I have very rarely failed to detect some loss of power. Poore[1] also insists that definite slight weakness of certain muscles of the hand is not uncommon. (iii.) Some discomfort is always experienced, and this sometimes amounts to actual pain. This pain, moreover, has a great tendency to spread. In some cases the pain is the most pronounced feature of the case, and is accompanied by tenderness of the nerve trunks, and tender points elsewhere (occupation neuralgia). "Pins and needles" and other subjective symptoms are by no means uncommon, but I am not aware that anæsthesia is ever observed. (iv.) Atrophy is said to be rare, but a careful comparison of the interossei and other muscles of both hands in several cases has convinced me that it does sometimes occur. Hypertrophy is much more frequent; it usually follows cases in which spasm is the leading feature. (v.) Electric irritability is slightly increased in the earlier stages and slightly diminished in the latter to both faradism and galvanism, in the muscles and the nerves (Gowers). (vi.) Twitchings and clonic spasms are only occasionally observed; but tremor and unsteadiness are common.

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[1] Vivian Poore, *The Practitioner*, 1878, and Med. Chi. Trans., vol. 61.

Course and Prognosis. The advent is very gradual, and the course prolonged over many months or years, depending on the continuance of the cause. The prognosis depends on (i.) the duration of the affection and stage when the treatment was commenced; (ii.) the means of the patient to cease the occupation; and (iii.) his intelligence in grasping the principles of treatment.

The diagnosis, by reason of its association with the occupation, is not usually difficult, but there are several organic and functional disorders which may be mistaken for writers' cramp at their outset. Brachial neuritis (*q.v.*), lead palsy, and even disseminated sclerosis may be mistaken for it, but in these careful inquiry shows that even from the first the symptoms were not solely determined by the act of writing.

Treatment. The disease is more frequent in nervous, sensitive persons, especially when suffering from a general "lowered tone," or malnutrition, anxiety, or grief. There is also a distinct tendency in persons of a neurotic family history. The fact that there is but one determining cause should render the treatment comparatively simple, but, as a matter of fact it is very difficult because the malady is chiefly found in those whose daily bread depends upon the performance of a certain muscular movement. Careful re-education can, however, accomplish a great deal, and particularly the adoption of a freer and larger style of writing, *by holding the pen more loosely and in a different way.* The patient should learn to hold the pen between the first and second fingers with the *back* of the hand against the paper and practise writing a series of large sloping M's. A certain amount of rest, at any rate at the outset, is indispensable. Meantime, the patient should begin learning to write with his left hand. Sedatives are of service, especially in neuralgic forms, such as chloral, bromide, small doses of morphia, physostygma, Indian hemp, belladonna, or atropine (one-hundredth of a grain hypodermically two or three times a week). Nervine tonics are recommended, and even strychnine, but I have often observed this remedy do more harm than good. Faradism also does harm, but voltaic electricity has often seemed to me particularly beneficial, especially when combined with massage. Gymnastic exercises are strongly advocated by some. One remedy, cod-liver oil, has rendered great service, and some of the most successful cases I have seen have been treated by a combined method of moderating the amount and improving the style of the writing, with small doses of bromide and cod-liver oil. In many cases dyspepsia or a rheumatic or gouty taint is present which should be met by appropriate measures.

OTHER OCCUPATION NEUROSES resembling the foregoing are found among telegraphists, drapers (in using scissors), cigarette rollers, violin players, piano players, typewriters; any occupation in short which necessitates the constant repetition of one particular movement.

IV. **Cramp** is a tonic muscular spasm occurring in one or more muscles of a limb. It is a troublesome symptom of not infrequent occurrence in persons in apparent good health. It is most apt to come on at night. A muscular cramp of great severity may seize one or all the limbs, and even the respiratory and trunk muscles of a swimmer, and prove very deadly unless aid is close at hand. It is apt to come on in those whose muscles are exhausted, but a low temperature of the water plays some part. Some persons seem prone to be affected by cramp throughout life on slight causes such as lying with a limb in a strained position. Others only suffer from it when their digestion or general health is out of order. The best remedy is to get out of bed and gently move and rub the limb. Bromide and chloral hydrate will generally prevent its occurrence, and any lithæmia, gouty or rheumatic tendencies should be corrected. Cramp is a frequent premonitory symptom in peripheral neuritis and phlebitis. It is also met with in subjects of Bright's disease and gout, and cramp of the legs is a painful symptom in the first stage of cholera.

§ **579.** V. **Tetanus** is a severe disease characterised by paroxysms of tonic and sometimes clonic spasms, due to the inoculation into a scratch or wound of a specific microbe whose chief habitat is in the earth.

Symptoms. (1) Within a few days after the injury the patient complains of stiffness of the jaw and back of the neck. (2) Very soon these muscles become rigid; the condition of the jaw is known as trismus, or lock-jaw, in which the teeth cannot be separated more than $\frac{1}{4}$ inch. This *tonic* rigidity affects all the muscles of the trunk, and in a less degree, of the extremities. The back is rigid, sometimes arched in the position of *opisthotonos*, in which only the head and buttocks rest on the bed. Or there may be flexion to one side—*pleurothotonos*, or bending forward of the body—*emprosthotonos*. The angles of the mouth are drawn down, and the eyebrows are elevated—*risus sardonicus*. (3) Clonic spasms supervene, in which the already rigid muscles become more contracted, with agonising pain. The slightest touch may cause clonic spasms. In severe cases these spasms become more frequent, leading to death from involvement of the glottis or respiratory muscles. (4) The temperature may be normal or slightly raised throughout, and may rise to 108 just before death. There is often retention of urine. The mind is clear till quite the end. A *variety* due to head wounds is described, with paralysis of the facial muscles and difficulty in swallowing.

Diagnosis. In hydrophobia the spasm is at first clonic : it affects chiefly the muscles of respiration and deglutition, and there is more mental agitation than in tetanus. In strychnia poisoning in the intervals between the spasms the muscles relax, and the spasms involve the extremities to a greater degree. In spinal meningitis there is a temperature, and there is no trismus. Tetany does not resemble tetanus. In hysterical opisthotonos there are other evidences of hysteria. Trismus is caused also by disease of the pons and acute bulbar paralysis, and in association with periostitis of the jaw, disease of the temporo-maxillary joint, or other local irritation, but the course of the disease serves to differentiate these from tetanus.

Prognosis. Death occurs in acute cases in one to twelve days from exhaustion or involvement of the glottis or respiratory muscles. In more chronic cases the spasms become less frequent and recovery ensues in 2 or more months. The mortality is very high, about 90 per cent.

Etiology. Tetanus is caused by a wound, however trivial, into which the tetanus bacillus has entered. The bacillus has its habitat in the earth.[1] Tetanus of the new-born is due to want of aseptic precautions in treating the navel.

Local *treatment* of the wound is necessary by cautery or antisepsis. For the spasms chloroform, bromides, chloral hydrate and other drugs are given, and recoveries have been reported after their use. The tetanus antitoxin has given good results in the sub-acute cases ; for acute cases unless given very promptly it is not so useful because the poison has combined with the nerve cells before the diagnosis is made and the antitoxin administered.

§ 580. VI. **Tetany** is a muscular stiffness occurring in paroxysms affecting mainly the ends of the four extremities.

The *symptoms* come on mostly in infancy in the form of a muscular spasm, or to be more correct, a paroxysmal stiffness affecting both the forearms, hands and feet. The attitude of the fingers compressed into a cone (the accoucheur's hand) has been emphasised, but it is unessential ; it is the attitude assumed by athetosis and many other tonic spasms. The paroxysms last from a few seconds to an hour or so, and in severe cases there is no intervening relaxation. In severe cases moreover all the muscles of the body are affected and there may even be opisthotonos. There is a good deal of neuro-muscular irritability to compression and to both forms of electricity, and if the nail be drawn down the face a wave of muscular contractility follows it. Many degrees of severity are seen, and the disease may last only 2 days or 2 or more months, recovery being the general but not invariable rule. The *diagnosis* is not difficult. In tetanus the spasm mainly affects the jaw, and there is a history of injury.

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[1] *The Lancet*, November 24, 1888.

Etiology. Tetany is most frequent in children the subjects of rickets or diarrhœa or other gastro-intestinal disturbance; but it is by no means confined to childhood. In adults it is particularly associated with dilatation of the stomach[1] or any other gastro-intestinal condition attended with fermentation. Cases have been noted in association with pregnancy, albuminuria and after removal of the thyroid. The disease is almost certainly due to a muscular toxin.

The *treatment* should be directed to the causal condition; bromide, chloral hydrate, and immersion in cold water (unlike Thomsen's disease) will relieve the spasm.

§ 581. VII. Hydrophobia is a contagious disease characterised by spasms of the muscles of deglutition and respiration and due to the inoculation of the saliva of an animal suffering from rabies.

Symptoms. (1) After an incubation stage, during which the patient presents no symptoms, which is generally about 6 weeks, never less than 12 days, and may even last as long as 12 to 18 months or more, there is an insidious onset of malaise with perhaps slight fever and sometimes tingling in the wound. (2) With or without premonitory symptoms paroxysms of painful spasms of the pharynx supervene, coming on at first with a slight stiffness and brought on by any attempt to swallow. (3) These spasms, at first clonic, become tonic, lasting ¼ to ½ hour at a time, and spread to the muscles of respiration and of the neck. The attacks produce excruciating pain and agony of mind. The mind is quite clear, but in the intervals there are prostration and general hyperæsthesia. (4) Paralysis ensues in 3 or 4 days' time, first of the muscles of the lower jaw, and death follows within a week from the onset.

Treatment. To destroy the virus at the seat of entrance suction immediately after the bite is heroic but efficacious. Cauterising the wound may be employed. The immunisation treatment of Pasteur is dealt with on p. 662. Narcotics, chloroform inhalation and chloral may be employed.

§ 582. VIII. Thomsen's disease (congenital myotonia) is a rare and obscure condition of universal muscular stiffness of indefinite duration aggravated by rest. Tension and stiffness of the limbs are experienced on first attempting to rise after resting, but they relax to some extent after continued movement. The muscles of the lower extremities are affected most, but those of the face, tongue, and eyes may be similarly involved. The stiffness is increased by cold. Sometimes there appears to be slight hypertrophy, never atrophy. The galvanic reactions vary, but faradism is never lost, and this leads to the idea that the disease is in the muscles, not in the nerves. The malady is noticed for the first time in youth and is very probably congenital. Several members of a family may be affected. The disease does not appear to shorten life. Warmth and continued activity relax the spasm to some extent.

IX. **Arthritic** rigidity is known by its being associated with some joint lesion, though the latter may be very slight. It affects both the extensors and flexors of the joint, though chiefly the flexors. I have usually noticed that it is increased during sleep.

There are **involuntary movements** *or shaking of the affected muscles, the range of the movements being either small* (TREMOR), *or large* (CLONIC SPASM.)

§ 583. **Tremors** and **clonic muscular spasms** form a very frequent and pronounced symptom in many different nerve lesions. For clinical purposes abnormal muscular movements may be divided into tremors or movements of small size, and clonic spasms or movements of larger range; paralysis agitans may be regarded as a type of the tremors, chorea as a type of the clonic spasms. The symptoms we are now considering must not be confused with generalised convulsions or fits (§ 591), nor with the uncertain movements of paralysed limbs or tabes dorsalis.

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[1] For a carefully studied fatal case of tetany following dilatation of the stomach, see J. S. McKendrick, *The Lancet*, Sept. 24, 1898.

CLINICAL INVESTIGATION. The more important points to be observed about cases of tremor or clonic spasm (in addition to their size or range as just mentioned) are—1. Their *distribution*, which may be localised to one limb as in hysteria, or generalised as in alcoholic tremors.

2. The *rhythm* or regularity of the tremor should be noted; for example, it is regular in paralysis agitans, senile tremor, disseminated sclerosis, Graves' disease, small hysterical tremors, alcoholic and metallic tremors, whereas it is irregular in the various forms of chorea, and in some functional conditions.

3. It should always be ascertained whether the tremor is present when the limb is *resting* on the bed or table, or only when the muscles are in action. Some kinds of tremor come on only during muscular action, as in disseminated sclerosis—this is known as *intention* tremor. Other kinds of tremor, such as paralysis agitans, persist during muscular rest, *e.g.*, when the limb is laid on a table. It should, however, be borne in mind that all kinds of tremor tend to be increased when the muscles are thrown into action, or when the patient's attention is directed to them, or during emotional states.

4. The *mode of advent* does not afford much valuable information. It is apt to be sudden in all forms of hysterical and functional spasm, but in most of the remainder it is gradual.

5. The *age* of the patient often gives us an important clue, for among the various causes paralysis agitans and senile tremor are uniformly found among the aged; whereas alcoholic tremor, metallic tremor, and paralytic dementia invariably affect persons in the middle decades of life; and finally hysterical tremor, chorea, disseminated sclerosis, and habit spasm are mostly met with in the comparatively young.

6. *Causation and pathology.* All tremors and clonic spasms may arise under one of three pathological conditions—(1) some organic lesion of the nervous system, such as disseminated sclerosis; (2) idiopathic or toxæmic causes such as general debility, anæmia, alcohol, hysteria, syphilis, rheumatism, or other toxic conditions; or (3) reflex irritation such as may be seen in the case of facial spasm or habit spasm, when the tremor may disappear on the removal of a diseased tooth, or correction of the refraction of the eye, or, as in a case I have seen, remedy of disease of the pharynx.

A study of the organic cases throws much light on the pathology of tremors and clonic spasm, for we find that they are always due either to partial destruction or imperfect recovery. The greater frequency of post-hemiplegic clonic spasms (1) after embolic lesions, which are less destructive than hæmorrhage, (2) after hemiplegia associated with hemianæsthesia, which indicates a lesion far back in the internal capsule, and (3) after the hemiplegia of children, in whom the recuperative power is so much greater, all point to this conclusion. Turning to other diseases we see that insular or disseminated sclerosis is attended by an incomplete destruction of the motor tract. Paralysis agitans again is evidently one of the senile degenerations, which is very, very gradual, and therefore for a long while incomplete. Later on in this disease, when the motor fibres are more completely destroyed, the tremors cease and paralysis with tonic rigidity ensues. Finally it may be seen what a very large proportion of the causes of tremor and clonic spasm are of toxæmic, idiopathic, or functional origin, and it is in just such functional causes where one would find only partial destruction (or partial recovery) of the motor tract.

Classification. It will be convenient to consider first the diseases in which tremors occur (such as paralysis agitans), and later the causes of clonic spasms (such as chorea). But this division must not be taken too absolutely, for those which are commonly small are occasionally apt to be large, and *vice versâ.*

TREMORS.

Partial degenerations.

I. Paralysis agitans.
II. Senile tremor.
III. Disseminated sclerosis.

Functional.

IV. Hysterical trembling.

Toxæmic.

V. General paralysis of the Insane.
VI. Alcoholic tremor.
VII. Metallic tremor.
VIII. Other toxic conditions.
IX. Nervous or neurasthenic tremor, and Graves' disease.

Organic.

X. Organic diseases in which tremor is not always a prominent feature :—
  (1) Post-paralytic tremor.
  (2) Amyotrophic paralysis.
  (3) Lateral sclerosis, Friedreich's disease, and other degenerative conditions.

CLONIC SPASMS.

Generalised.

I. Chorea.
II. Hysterical spasms.
III. Myoclonus multiplex.

Localised.

IV. Habit spasm.
V. Facial spasm.
VI. Spasmodic torticollis.
VII. Other localised spasms.

Organic.

VIII. Post-paralytic clonic spasms.

§ **584.** I. **Paralysis agitans** (syns., shaking palsy, Parkinson's disease). The onset is extremely gradual, and the three cardinal symptoms are :—(i.) the tremor, which is moderate in size, rhythmical, and of general distribution, though excepting the head and neck. It continues when the limbs are supported. Not infrequently it predominates on one side of the body, or in the arms or the legs, and the onset is often accompanied by "rheumatic" pains, facts which point to a degenerative lesion in the peripheral nerves.[1] (ii.) The muscles gradually become stiff, and the attitude and aspect of the patient (Fig. 4, p. 29) are very characteristic. The head appears to be fixed, and is bent a little forward ; and in walking the patient has a tendency to fall forward (festination). The patient's face appears like an expressionless mask, with the eyes always looking forwards. (iii.) The patient is nearly always over 40 years of age, and, in 65 per cent. of the cases, of the male sex. (iv.) The other and less important points for differential purposes are, progressive weakness, which gradually involves all the limbs ; the speech, which is monotonous and slow ; and various subjective sensations, such as restlessness and a continuous desire to be moved, if in bed. The intellect is preserved, and though the disease lasts for many years, it does not usually shorten life. No complaint is made of vertigo or nystagmus. Towards the end of life the tremor disappears, and is replaced by paralysis and organic contracture.

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[1] In one case Dr. Robert Maguire and the author found after death degenerative lesions in all the chief peripheral nerves.

II. **A senile tremor** is described by some, but in the course of a large infirmary experience I was rarely able satisfactorily to differentiate paralysis agitans from senile tremor. However, in the latter the tremor always involves the head and neck, being indeed sometimes most marked in or limited to this situation, and rigidity and paresis are less marked.

§ 585. III. **Disseminated sclerosis.** Islets of sclerosis in the spinal cord were described by Cruveilhier as an anatomical condition, but the clinical symptoms attaching to the condition were not described until Professor J. M. Charcot studied this condition clinically, and gave to it the above appropriate name. There are three types of the disease recognised, the spinal, cerebral, and cerebro-spinal, according to the part chiefly involved. The three characteristic symptoms of this disease are (i.) the tremor, which is very rhythmical, rather larger than paralysis agitans, and occurs only when the muscles are in action, ceasing when the limb is supported ; this is, indeed, the most typical of "intention tremors." It involves all the muscles, including those of the head and the tongue. The intention character differentiates the tremor from paralysis agitans, and it may be distinguished from chorea by the fact that the arm goes straight to its goal, as for instance in feeding, whereas in chorea it takes a zig-zag course. (ii.) The patient is usually under 30 (the earliest case I have observed was 16 years old) ; it rarely commences in persons over 45 or 50 years of age. The sexes are equally affected. (iii.) The speech is characteristic, being slow and syllabic, *i.e.*, pausing between the syllables of a word, and clipping the labial consonants. There is a torpid condition of the intellect, especially in those cases where the brain is also involved. (iv.) Vertigo is also an important and early symptom, occurring in something like 70 per cent. of the cases. (v.) Nystagmus is another constant symptom, and it is often accompanied by primary optic atrophy (white atrophy). (vi.) Various other symptoms may arise, according to the position of the patches of sclerosis. Thus, if the lateral columns be much involved, we get spastic symptoms ; if the posterior columns, tabetic, and so on. In an analysis of 50 cases, Dr. F. S. Palmer[1] found that weakness, especially of one limb only, was the first symptom noticed in the disease in 15 cases. The patellar reflexes are usually increased, but they may be normal or diminished ; their absence is serious, as denoting involvement of the grey matter. Various sensory disturbances may also be observed. (vii.) The course of disseminated sclerosis is prolonged ; it may last from 2 to 10 years, being longest in the purely spinal form, shortest in the cerebro-spinal form.

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[1] "Early Manifestations of Insular Sclerosis," *Med. Press and Circular*, Sept. 7, 1904.

IV. **Hysterical tremor** is a very frequent manifestation of that diathesis. It may be small and regular or large and choreïform (see below).

V. **General Paralysis of the Insane** has been fully described in mental disorders. Here the tremor is very fine, regular, and vibratile, and it tends to affect the lips and tongue more than any other parts ; indeed, at first these alone may be involved. It is accompanied by more or less general weakness and by characteristic mental alteration.

VI. **Alcoholic tremor** is one of the most constant evidences of chronic alcoholism. It is specially noticeable in the hands, is small and vibratile, rhythmical, dependent on muscular action (intention tremor), and worst in the morning. There is also a history of dyspepsia with morning vomiting, insomnia, and other evidences of chronic alcoholism.

VII. **Metallic tremor.** Mercury, lead, and zinc, especially when introduced into the system in the form of vapour, or in small doses for a considerable length of time, not infrequently produce tremor. It is small, rhythmical, and only apparent when the muscles are in action. It is also characterised by the other signs of poisoning by the respective metals. Mercurial tremors occur, for the most part amongst thermometer-makers, and water-gilders in the manufacture of mirrors. Lead tremors occur largely amongst the Cornish and Cumberland lead-miners, and zinc poisoning amongst brass-founders, who are exposed to the fumes of oxide or oxychloride of zinc.

VIII. Other **toxæmic conditions** are also attended by tremor, such, for instance, as diabetes, ague, influenza, and pyrexial states. Here the tremors are small and rhythmical, and a history of the cause is easily made out. **Graves' disease** is mostly attended by a fibrillary tremor, and in such cases inquiry should be made for thyroid enlargement, ocular prominence, cardio-vascular phenomena, and other neurasthenic symptoms, the tremor being really one of these.

IX. **Nervous tremor** arises from either debility of the muscular or nervous system, and is seen in its most typical form in cases of neurasthenia (§ 518), or general debility from any cause, under which headings the other characters of the various causal conditions are described. Tremor may also be present in **occupation neuroses** (§ 578).

X. There are **organic diseases** also in which tremor occurs, but it is usually accompanied by other and more prominent symptoms.

In many cases of *amyotrophic paralysis*, the muscles are affected with a fine fibrillary tremor.

Tumour of the brain may be accompanied by muscular movements, especially (i.) when the tumour presses on, without actually destroying, some part of the motor tract, *e.g.*, in children who are the subjects of a tuberculous tumour near the crura or the posterior part of the internal capsule, there is tremor exactly like that of disseminated sclerosis. (ii.) If it be situated in the parietal region or near the optic thalamus, it usually takes the form of spastic athetoid movements. (iii.) Cerebellar lesions may be associated with spasmodic movements of the neck muscles on one or other side.

Lateral sclerosis often gives rise to tremor of the legs on walking or any muscular movement.

Friedreich's disease is chiefly characterised by tremor or by disorderly movements.

§ **586. I. Chorea** (St. Vitus's dance ; Sydenham's chorea) is a disorder of the nervous system generally occurring in childhood, characterised by irregular awkward movements of the limbs and a tendency to cardiac valvular disease, running a more or less definite course usually towards spontaneous recovery. It was first described by Sydenham in the seventeenth century.

(1) The movements usually partake more of the character of gesticulations or exaggerations of normal movements than of tremors or even clonic spasms. In carrying a spoon to the mouth, the hand does not reach its goal, as it does in disseminated sclerosis. The movements may involve all the muscles of the body, even those of deglutition, the face, the tongue and respiration, including the diaphragm. They are sometimes hemiplegic in distribution, and may predominate in the upper extremity, or the face. The movements cease during sleep. The deep reflexes vary. (2) A certain amount of hemiparesis may accompany or alternate with the movements, and the chief symptom for which many patients are brought is because they drop things or fall down. Sometimes they are brought for restlessness at school. Very severe cases may develop maniacal symptoms. Eruptions on the skin (erythema, herpes zoster, purpura and subcutaneous nodules) have been observed. (3) It is also accompanied, in a large proportion of recurrent cases, by valvular disease, which is indistinguishable from that of rheumatic origin.

The *diagnosis* from habit spasm is occasionally difficult, but the latter is more persistent in duration, more limited in distribution, and the involuntary movements always affect the same set of muscles. It may also have to be diagnosed from various other clonic spasms mentioned below.

The *prognosis* is usually favourable, and tends to spontaneous cure in the course of 1 to 3 months, though in about one-third of the cases permanent cardiac disease remains, and 20 per cent. of all cases of chorea get rheumatism within six years.[1] The severity of the attack depends a good deal upon the age of the patient. Over the age of puberty the disease assumes a much graver aspect, and is very apt to be recurrent, and when it complicates pregnancy in young women the mortality, according to Dr. Robert Barnes,[2] amounts to 30 per cent. Great severity of movement, recurrence of attack, and maniacal symptoms (chorea insaniens) are always grave.

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[1] Dr. F. E. Batten, *The Lancet*, November 5, 1898.

[2] St. Thomas's Hospital Reports, about 1870.

Etiology. The disease is essentially one of childhood and is three times as frequent in the female. There is a special liability for chorea to follow rheumatism, quinsy, scarlatina, and to a less extent other infective disorders. For many years I have taught that chorea was microbic in origin and pointed out in proof (1) the practical limitation of the disease to childhood; (2) its more or less definite course and tendency to spontaneous recovery; (3) the marked tendency to endocarditis, and (4) the spread of the disease to other children by so-called "imitation." In 1903 Dr. J. F. Poynton revealed the rheumatic diplococcus in cases of chorea, and the disease is now coming to be regarded as microbic. As in rheumatism, there is a marked tendency to recurrence and it is predisposed to by many of the same conditions as the acute specific fevers. Fright or other sudden emotion may determine an attack.

Treatment. Salicylates as first introduced by Dr. David B. Lees, administered in the same way as in rheumatism, are almost as successful in chorea. Aspirin and quinine have been tried. The patient must be taken from school. The movements if violent may need bromides and large doses of chloral (gr. x. every 2 hours if awake) or trional, and a water-bed is desirable. Arsenic in gradually increasing doses is the best tonic and may alone be sufficient to cure mild cases. A wet pack and other methods for the application of heat or cold are excellent means of treating severe cases (see § 590).

Ia. **Huntingdon's Chorea** (Chronic chorea; Hereditary chorea) is characterised by irregular gesticulatory movements, coming on gradually between the 30th and 40th year and lasting for the rest of life. The gait is irregular and swaying, with sudden stoppages, the speech is affected, and there is mental impairment leading to dementia. This disease runs in families. A similar affection coming on in the aged, without hereditary cause, is known as *senile chorea.* Rhythmical and other forms of chorea are mentioned below. It is unfortunate that the term chorea has been applied to these various conditions which are totally distinct from Sydenham's chorea. *Henoch's chorea electrica* is probably identical with paramyoclonus multiplex (below).

II. **Hysterical** tremor and spasms are of different kinds; they may be generalised, like chorea, or localised to one or another situation; and they are aggravated by but not dependent on voluntary muscular action. They are all characterised by—(1) their sudden access, generally after some emotional shock; (2) they vary in size, rhythm, and even in position from hour to hour and day to day; (3) they have a tendency to predominate in the head, neck, or arm, and (4) they occur in a characteristic age and sex. A typical case of this sort was that of a young lady, æt. 24, whom I saw lately, who had suddenly developed choreïform movements in the right upper extremity, while under the unusual influences of a long stay at her fiancé's home. The disorder consisted of a continuous regular tremor of the arm, interspersed with attacks of large irregular clonic spasms. The knee-jerks were so much exaggerated that the patient almost jumped out of the chair. Another class of hysterical spasm has been appropriately named chorée rhythmée by Charcot, in which condition the movements are distinctly choreïform, more or less generalised, and differing from ordinary chorea, first, by their coming on in attacks, or at any rate being liable to severe exacerbations; and secondly, by having a tendency to a certain degree of regularity. In one case of this kind which I observed,[1] these attacks could be started by pressure on the mamma, and they could be stopped by steady pressure in the ovarian region. In addition to these two forms, which might be called localised hysterical spasms and chorée rhythmée respectively, there are a large number of other rarer kinds, which may be provisionally placed in the hysterical group, though their precise relation to hysteria has not been made out. For instance, *saltatory spasm* (jumping chorea) is a rare condition of clonic spasms, affecting principally the lower extremities, which are usually in a state of more or less rigidity, and subject to violent extensor spasms whenever the soles of the feet are touched, or when the patient is placed upon the feet. The hands and arms are usually free. Hammering and dancing chorea have also been described by Charcot[2] and others.

III. **Myoclonus multiplex** is a very rare condition described by Friedreich.[3] Judging by the few cases that have been recorded, this condition may be provisionally described as a disease consisting of sudden very small shock-like clonic muscular spasms, not usually sufficient to produce tremor of the limbs, bilateral, affecting mainly the proximal segments of the 4 extremities; occurring mostly in males, and running a chronic course. Spasms of larger range may occur. It is probably due to some congenital or hereditary defect, though it may not be revealed for several years after birth. The most curious part of this strange disorder is the exemption of the wrists, ankles, hands, and feet. It is usually relieved by fairly strong galvanic currents, but is apt to relapse. The only case that I have seen had lasted for nearly 30 years. Much confusion exists as to the identity of the disease.

§ **587.** IV. **Tic** or **Habit Spasm** is the first cousin of chorea. It is, in fact, a localised choreïform movement, and it is often difficult to distinguish between the two diseases. It practically always starts in childhood, mostly in girls between 7 and 14, and is indeed an exaggeration of the normal restlessness of this age. It consists of sudden quick muscular twitchings, say of the eyelids, face (causing grimaces), shoulder or arm, differing from chorea in (i.) being always limited to one place, (ii.) performing the same action, and (iii.) running an indefinite course. The face and arms are the most frequent situations, but the muscles of respiration or any other part may be involved, and respiratory sniffs or grunts are not infrequent. It is always worst when the child is nervous or attempts to restrain the movements; and parents should be cautioned not to scold the child for the habit. There is frequently a history of neurosis in the family, and habit spasm may certainly arise by the "imitation" of other children. Arsenic is of considerable value, but other medicines are not of much use. Removal from the surroundings under which the disease arose is the most efficient remedy.

Impulsive tic is a condition which has been more particularly studied by Gilles de la Tourette, allied to habit spasm and like it arising for the first time in children, but not confined to them. It consists of explosive muscular movements, usually of the face or arms, but in violent cases all over the body. Explosive sounds accompany these movements, either barking or inarticulate sounds, or the constant repetition of one word (echolalia), or some obscene or swearing word (coprolalia). The condition often occurs in association with some mental defect.

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[1] *Clinical Journal*, October 19, 1898.

[2] "Leçons Cliniques sur les Maladies du Système Nerveux."

[3] Virchow's Archiv. Bd. lxxxvi., p. 421, 1881.

§ 588. V. **Clonic facial spasm** (syns. spasmodic tic, convulsive tic, tic non-douloureux, mimic spasm) is the term employed for a condition of persistent clonic spasm of the muscles of the face. Clonic facial spasms may arise under 3 different conditions—organic lesion, reflex irritation, and idiopathic or constitutional causes. The favourite age for facial spasm is between 30 and 60.

Organic lesion may give rise to facial spasm in the same way as it gives rise to facial paralysis, and this cause is known either by the presence or a past history of paralysis. However, if spasm be present, it is an indication either that the destruction of the facial nerve tracts was incomplete, or else that partial recovery had taken place. In this way tumours, such as sarcoma or aneurism of the vertebral artery, pressing on without entirely destroying the facial nerve, give rise to facial spasm. A fall on the head, or a cortical injury at birth may act similarly. Diseases of the bones (syphilitic or tuberculous) through which the nerve passes may also be suspected; and all the structures beside which the nerve passes should be carefully and thoroughly investigated. If the lesion be cerebral, the spasm is more general, and is apt to involve muscles physiologically associated, perhaps on opposite sides. Thus, if both corrugators and the muscles on one cheek only be involved, a cortical lesion is indicated. If it be the nucleus or the nerve trunk that is affected, we then find that there are or have been the paresis and electrical changes characteristic of this condition.

Reflex irritation may be in operation, especially of the 5th nerve. The teeth should be examined, a history of injury to the 5th inquired for, pharyngeal adenoids removed, and the refraction should be tested. *Idiopathic causes*, such as grief or other emotion, debility, anæmia, the climacteric, hysteria, gout, rheumatism, syphilis, or tubercle, may act as contributory causes.

Facial spasm of *organic* origin may be *diagnosed* from habit spasm by its persistence, the age of the patient, the surrounding circumstances, and electrical changes (if any). But I know of no means of distinguishing *non-organic* facial spasm from habit spasm in the face though all the books describe them separately.

Course and treatment. Facial spasm, as above described, is generally a very chronic and progressive condition, resisting all our efforts excepting in those cases where the cause is removable, and the causes should be thoroughly investigated on the lines above given. Weak galvanism of the affected nerve may be tried. To palliate the spasm bromides, gelsemium, cimicifuga, antipyrin, chloral, and in extremes morphia and other nerve sedatives may be employed.

§ 589. VI. **Torticollis** (wry-neck) is a spasm of the muscles on one side of the neck. Wry-neck is of two kinds, the congenital, due probably to injury at birth, and the acquired, which may appear at any age. In the former condition the spasm is tonic; in the latter the spasm is chiefly clonic, but in long-standing cases there may be some tonic spasm as well. (a) *Congenital* wry-neck is due to a contraction of the sterno-mastoid of one side, rarely both. The chin is projected upwards and to the opposite side. It may not be noticed by the parents till the child is several years old. It is generally associated with facial asymmetry, as pointed out by Sir Samuel Wilks. The only remedy is tenotomy. (b) *Acquired* torticollis consists of a slow clonic spasm, recurring every few minutes, associated in long-standing cases with a certain amount of tonic spasm which, when the sterno-mastoid is involved (as is usual), draws the head to the opposite side. In about half the cases the trapezius is associated in the spasm; in other cases the splenius, scalenus, and platysma may also be involved. The condition is very intractable.

Causes (compare also the remarks on facial spasm which apply here) and *treatment.* The causes of torticollis are often obscure. The acquired form very commonly appears to be of hysterical origin. I have met with cases coming on after injury to the back of the head, and in some cases relief has been obtained by remedying a gouty or rheumatic habit of body. Like facial spasm (*q.v.*), wry-neck probably arises under 3 different conditions—organic lesion, reflex irritation, and idiopathic causes, and treatment should be directed to these. Many drugs have been tried without much benefit. Galvanism of the spinal accessory nerve regularly applied twice daily has in one instance produced permanent relief. Surgical means (stretching, division or excision of the nerve, or division of the muscle and resection of the posterior branches of the upper cervical nerves) have been tried, but as far as I am aware, without much success.

VII. Localised clonic spasm may affect almost any voluntary muscle or group of muscles in the body. The case should be investigated on the lines just indicated under facial spasm: a large proportion are hysterical. In clonic spasm of the *diaphragm* very curious respiratory spasms and grunts occur; hysteria and possible reflex causes of irritation in the stomach should be remembered. It is also seen with organic cerebral disease. Clonic *masticatory* spasms may arise in paralysis agitans and old age, and sometimes in hysteria. Clonic spasm of the *respiratory muscles* causing forced respiration is almost always due to hysteria. *Nutatory* or nodding spasms occur in children at the time of dentition, or as a form of epilepsy, sometimes associated with nystagmus. In women it is usually due to hysteria. *Muscular twitchings* or startings may arise when the muscles are over-fatigued, or in the half-waking state in various conditions attended by constitutional debility (*q.v.*). They are an occasional symptom of neurasthenia, and twitchings of the same kind affecting the legs are also one of the earlier symptoms of peripheral neuritis.

§ 590. VIII. Post-paralytic clonic spasms. The fact that hemiplegia and paraplegia of organic origin is gradually succeeded by tonic rigidity and other evidences of lateral sclerosis has already been referred to, but occasionally we also get clonic movements of various kinds supervening sooner or later, especially when the paralysis has occurred in early life.[1] There are many different kinds of clonic spasm, but practically they come under 3 types, which, in order of frequency, are (a) a slow mobile spasm called *athetosis*, (b) movements which are more or less *rhythmical*, and (c) irregular choreïform movements not inappropriately called *post-hemiplegic chorea.* They are all characterised by (1) having the same distribution (generally hemiplegic) as that of the preceding paralysis, though in cases of hemiplegia the arm is always more affected than the leg. (2) The presence of the other symptoms of lateral sclerosis (§ 555). All of these movements are relatively rare in adults, but in such cases they are more frequent after hemiplegia due to an embolic lesion than after a hæmorrhagic one, and also after lesions which are associated with hemianæsthesia. The jerkings of the legs quite early in cases of paraplegia due to a complete transverse lesion with a fairly extensive area of healthy cord below, are caused not by descending sclerosis but, as most believe, by the cutting off of inhibitory influences from the brain.

The *prognosis of tremors and clonic spasms* in general terms is much more favourable than that of hemi- or paraplegia, a fact which is in keeping with the remarks on pathology in § 583. The prognosis of some has been dealt with in detail. (1) In none of the above conditions is there as a rule any immediate danger to life, excepting in certain cases of chorea and paralytic dementia. (2) There are but 3 of the above maladies which tend progressively to a lethal termination, viz., disseminated sclerosis, paralytic dementia, and progressive muscular atrophy. Disseminated sclerosis lasts for an average of 5 or 6 years, being shortest in the cerebro-spinal form and longest in the spinal form, the cerebral form occupying an intermediate position. In all four of these, the course rarely lasts longer than 10 years, and in acute cases only about a year or so. (3) Another group of the above diseases endure for a lifetime, though without materially shortening it; viz., paralysis agitans, senile tremor, lateral sclerosis, many cases of facial and neck spasm, and some of the rarer group allied to hysteria. Nevertheless, some of these can be ameliorated. (4) Many of them progress towards spontaneous recovery, *e.g.*, chorea, which is perhaps the most frequent clonic spasm met with, hysterical and nervous tremors, habit spasm, and many cases of facial spasm. (5) Many of the above are curable, chiefly by removing the cause, for example, in alcoholic, metallic, and other toxic tremors, Graves' disease, and the great majority of those which depend on reflex and idiopathic causes.

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[1] This is one of the facts supporting the belief that tremors and clonic spasms of organic origin are due to partial destruction or partial recovery of a motor nerve tract. It is the recuperative power of childhood which leads to partial recovery.

Indications for the *treatment of muscular tremor and spasm.* For the rational treatment, the reader should refer to the brief reference to the pathology (§ 583), giving the 3 pathological causes on which the cases may depend. The indications for treatment are four-fold. (1) To restore the partially damaged motor fibres or otherwise remove the cause. Iodide of potassium is of use, not only in cases of known syphilitic origin, to promote absorption, but also to eliminate toxic agents, such as lead, mercury, etc. (2) Careful investigation should always be made for any reflex irritation, such, for instance, as any uterine, stomach, and other visceral trouble in cases of hysterical spasm. (3) The third indication is to remedy any constitutional or general defect, such as anæmia, rheumatism, debility, over-work, or over-worry. (4) If the cause cannot be ascertained or eradicated, we can nevertheless, in many cases, alleviate the tremor or spasm by appropriate means; thus, hyoscyamus and its alkaloid (hyoscine hydrobromate, gr. $\frac{1}{200}$ to $\frac{1}{50}$ ter die) are very valuable in this respect, and many instances could be quoted of considerable relief in cases of paralysis agitans, senile tremor, disseminated sclerosis, etc. Cannabis Indica, physostigma, conium (succus ʒss, increased to ℥ss.) may also be tried. Opium and morphia are permissible in some severe cases, but, as a rule, the relief is only temporary. Bromides are undoubtedly of great value in neurasthenic and other nervous tremors, hysterical tremors and spasms, paralytic dementia and chorea. Warm baths, Turkish baths, and the application of heat is an agent for the relief of tremor and spasm which is too often neglected. Hot water internally and externally is the best remedy I know of for the relief of spasm of unstriped muscular fibres (such as spasmodic dysmenorrhœa) and the hot pack[1] or hot bath as mentioned before acts wonderfully in cases of chorea. The very simplicity of this remedy—the application of heat—renders it all the more valuable. In cases where the paralysis is a leading feature, strychnine may be given, though, as a rule, this remedy is contra-indicated in nearly all clonic spasms. Tremors and spasms of hysterical origin must be treated on general lines, as in other cases. Cold douches are useful for functional spasms. Localised muscular spasms are also treated by massage and electricity, the anodal pole being placed on the affected site.

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[1] A thick blanket should be thoroughly wetted in a pail of boiling water, wrung out, and rolled up tight, and then the patient rolled in it, afterwards in another dry one, and left for half an hour to perspire. It is often advisable to promote diaphoresis by a dose of liquor ammoniæ acetatis, for the benefit derivable is much less unless perspiration ensues.

The patient has **attacks** *of* **clonic and tonic spasms** *associated with more or less* DISTURBANCE OF CONSCIOUSNESS the case is one of CONVULSIONS.

§ 591. **Convulsions** are sudden, violent, clonic and sometimes tonic spasms affecting the greater part or the whole of the body, usually accompanied by some disturbance of consciousness.

The most common cause of convulsions is idiopathic epilepsy, and convulsions arising from other causes may so closely resemble epilepsy that they are often described as epileptiform convulsions. Eclampsia is an old term used for epileptiform convulsions. The following are the causes of convulsions—

Functional.

I. Idiopathic epilepsy.
II. Hysterical convulsions.

Organic lesions.

III. Intracranial syphilis.
IV. Gross lesions of an irritative nature, such as—(1) Intracranial tumour (Jacksonian epilepsy). (2) Hæmorrhage and embolism. (3) Chronic degenerations of the nervous system. (4) Acute meningitis.

Toxic causes.

V. Uræmia; diabetes; cholæmia.
VI. Puerperal eclampsia.
VII. Various other toxæmic conditions, such as—(1) Alcohol: (2) lead: (3) drugs: (4) tetanus (tonic spasm); diabetes; malaria; acute specific fevers (in children).

Circulatory.

VIII. Cardio-vascular disorders, including chronic Bright's disease.

Reflex.

IX. Reflex causes.

Convulsions in infancy and childhood.

The chief points in the CLINICAL INVESTIGATION of a convulsive attack are—the age of the patient, the character of the attack, the state immediately preceding and following it, and the attendant conditions. The commonest causes of convulsive fits in every-day practice are epilepsy, intracranial syphilis and hysteria. Ask *first* the *age*, and *secondly*, whether the patient ever had an attack before. If the patient be under 12 months old, turn to infantile convulsions (§ 593). Between 1 and 10 years of age, embolism, hæmorrhage and many organic conditions may give rise to convulsions. Between 10 and 20 is the commonest time for idiopathic epilepsy to commence. If the patient be over 30 and has never had a fit before, syphilis should be suspected. Hysterical convulsions mostly affect the female sex between 15 and 25 and at the climacteric. If the patient be over 50, uræmia and apoplexy should be suspected. *Thirdly*, the *characters of the fit* afford very considerable aid in diagnosis. Thus (i.) the convulsions of major epilepsy and major hysteria are nearly always generalised: partial convulsions indicate usually a focal cortical lesion (Jacksonian epilepsy). (ii.) Unconsciousness is an invariable accompaniment of epilepsy, and is very usual with uræmia, cerebral hæmorrhage, and syphilis. On the other hand, consciousness is not completely obliterated in by far the greater number of cases of hysterical convulsions, and in a considerable number of limited cortical lesions. *Fourthly*, inquiry should be made as to whether the fit was preceded by an aura (epilepsy) and what is the condition immediately afterwards. *Fifthly*, the history and attendant symptoms should be examined in the usual way.

§ 592. I. **Idiopathic epilepsy** (Syn. Falling sickness) is a disease attended by sudden attacks of loss of consciousness, with or without convulsions, without any discoverable cause in the blood or in the brain. It occurs in two distinct clinical forms, *minor* epilepsy (petit mal), which consists simply of a transitory disturbance of consciousness and is therefore referred to in § 524, and *major* epilepsy (haut mal, grand mal) which consists of a convulsive seizure with loss of consciousness.

Symptoms. A complete epileptic fit has the following characters, though they are rarely all present in their entirety. (1) In some cases, during the previous 12 to 24 hours there may be *prodromata* consisting of headache, giddiness, jerkings of the limbs, or alteration of character or mood. In more than half the cases this stage is absent. (2) The fit in many cases is immediately preceded by an *aura* or warning, *i.e.*, a sensation lasting at most only a few seconds, which is of value as indicating the point of the cortex whence the cortical nerve-storm starts. Of these auræ there are four groups. *Sensory auræ* are most common, *e.g.*, "a wave passing over the body," numbness, flashes of light or of colour, or singing in the ears; *motor auræ, e.g.*, twitching of a muscle or a limb, rarely of the trunk, and in rare cases there is a "procursive aura," in which the patient runs forward and turns round and round; *psychical auræ, e.g.*, various strange thoughts or perhaps illusions; and, *somatic auræ, e.g.*, gastric discomfort, nausea, or fluttering in the stomach. Some form of aura is present, in my experience, in a little more than one-half of the cases of all forms of epilepsy. (3) *Loss of* *consciousness* is the pathognomonic and indispensable feature of *all forms* of idiopathic epilepsy. It succeeds the aura so quickly that the patient rarely has time to place himself out of danger before loss of consciousness is complete. (4) *Convulsions* supervene almost at the same moment as the unconsciousness. They are often ushered in with a scream, and in the classical form consist of a short stage of tonic convulsions lasting about 40 seconds, followed by a stage of clonic convulsions lasting 1 to 3 minutes. In the tonic stage the breath is held, the hands clenched, the back rigid, the legs extended, the pulse quick and may be imperceptible. The clonic movements which succeed soon involve the whole body, and are sometimes of great violence, consisting of rapid extension and flexion of the limbs, opening and shutting of eyes and jaws. The tongue is often bitten—a danger to avoid by thrusting a piece of wood, the handle of a pocket-knife, or something of the kind between the jaws. The pupils are dilated and the conjunctivæ insensitive. During the tonic stage respiration is stopped, and the patient becomes blue in the face (a diagnostic point of great value) ; and as the convulsions pass off the respiration becomes stertorous or snoring. Urine, fæces, and even semen may be voided. The saliva issues from the mouth as a frothy foam, sometimes blood-stained from injury of the tongue. (5) A stage of *stupor* or *drowsiness* succeeds the convulsions, and may last for some hours. This stage passes gradually into a deep and profound sleep. The temperature directly after the convulsions is said to be raised, sometimes as much as 4° or 5°. (6) In the *post-epileptic state*, after recovery, there may be aphasia or transient paresis, or the patient may perform automatic, irresponsible acts, dressing or undressing himself, or putting the property of others into his own pocket. Occasionally hallucinations, delusions, or active mania ensue, or the patient makes obscene remarks or commits acts of violence. Some patients are distinctly homicidal, rarely suicidal. One fact of considerable forensic import must be mentioned ; namely, the phenomena of the post-epileptic state are by no means proportionate to the severity of the seizure (major or minor) which they follow—often quite the reverse. After severe fits the patient is exhausted, after slighter attacks his faculties are often stimulated.

The *intervals* between the fits vary considerably; any time from a few days to many years. In the intervals the patient usually remains in fairly good health of mind and body until the next attack; indeed, it frequently happens that he professes himself in better health after an attack than before. *Epilepsy* has a close association with *insanity*. In a certain proportion of cases the fits are followed by weakmindedness, whether the case has been treated with bromides or not; occasionally by other forms of insanity. It appears to be the frequency, rather than the severity, of the fits, the age at which they commenced and the heredity that determine the occurrence of mental alteration. Thus, petit mal quite as often leads to mental trouble as does epilepsy major; some say more often. Again, epileptic fits may be followed (and sometimes even replaced) by mania of a most dangerous nature, in which the patient may (quite unconsciously) commit acts of the most brutal kind. Epilepsy and insanity appear to be closely related to each other by their hereditary origin; one child may be epileptic, another insane.

Varieties. The above is the classical form of epilepsy major, but every degree of severity may be found between this and epilepsy minor. Frequently one or other, or several of the above features may be wanting, but unconsciousness is the one constant feature, and in epilepsy minor it may be the only symptom present. Sometimes fits of various kinds may alternate in the same patient; but generally each patient has fits which conform to one, or at most to two varieties. The *status epilepticus* is a rare condition in which the patient has a series of fits occurring in very rapid succession for several hours or even days, consciousness not being regained in the intervals; the temperature may rise to 107°, and the condition is often fatal.

The *diagnosis* of epilepsy minor will be found in § 524. Epilepsy major may have to be diagnosed from any of the causes of convulsions (see list, § 591). Convulsions coming on for the first time after 30 are more probably *syphilitic* than idiopathic. In *feigned epilepsy* the pupils are not dilated, and they react normally to light, the conjunctivæ are sensitive, and the application of strong ammonia to the nostrils generally reveals the fraud; the absence of the cyanotic condition may also aid. The alleged fact that the patient will not hurt himself is not of very great assistance, for I have often seen patients do themselves serious injury if the motive for the fraud is sufficiently powerful.

Table of Diagnosis of Epilepsy.

	Major Epilepsy.	Hysterical Convulsions.	Feigned Epilepsy.
Preceded by	Characteristic aura.	Globus, or choking, etc.	A motive for fraud.
Mode of onset.	Sudden.	Often gradual.	*Carefully planned.*
Characters of convulsions.	Definite march. Tonic brief, followed by clonic movements. Cyanosis during tonic stage ; tongue may be bitten ; urine and fæces may be voided.	Tonic contractions, and rigidity often prolonged and recurrent. Clonic movements often purposive and irregular ; *tongue not bitten ;* urine and fæces rarely voided ; usually no cyanosis.	Not following the usual march ; *absence of cyanosis and asphyxia ;* urine and fæces not voided ; tongue not bitten.
Consciousness.	Lost.	Rarely quite lost.	Retained.
Eye symptoms.	Pupils dilated and irresponsive to light, conjunctivæ insensitive.	Pupils responsive to light ; conjunctivæ generally sensitive.	*Conjunctivæ sensitive ; pupils responsive to light.*
Duration.	Very rarely exceeds 4 or 5 minutes.	Generally exceeds 4 or 5 minutes, may last ½ hour, and liable to recur.	Indefinite.
Termination.	Stupor and drowsiness.	Sobbing, crying, laughter, or prostration.	Not followed by profound stupor.

Course and prognosis. If unchecked by treatment the fits recur throughout life, though with widely varying frequency. The frequency of the fits is the leading factor in the prognosis. Those in whom fits occur with moderate severity a few times per annum may remain well and clever, but when more frequent mental deterioration results, not, be it remembered, as a consequence of the administration of bromide, but as part of the disease. Indeed, in petit mal, on which bromide has least influence, mental symptoms ensue more often than in grand mal. A combination of the two forms is worse than either singly. Death may occur from an accident during the fit, but rarely or never from the disease.

Etiology. Both sexes are about equally affected. About 75 per cent. start the fits before the age of 14 ; idiopathic epilepsy very

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rarely begins after 20. Heredity is a potent factor, and in a large proportion there is a family history of epilepsy or other nervous ailments. Among the determining causes of a fit may be mentioned fright, excitement, head injuries, the menstrual period (many females have fits only at that time), alcoholic and sexual excess. Epileptic fits have been known to be associated reflexly with such causes as the irritation of a scar, disease of the eyeball, disease of the nasal mucous membrane, on the removal of which the fits ceased. My colleague, Mr. Work Dodd,[1] has shown, moreover, that in certain epileptics having a high degree of asthenopia, the fits become much less frequent or cease altogether when the error of refraction is accurately corrected and the asthenopia relieved.

It is worthy of note that Hughlings Jackson[2] believes that every epileptic patient has an epileptiginous zone or area, corresponding on the surface of the body to the centre in the brain whence the cortical discharge starts, but in actual practice it is rarely possible to discover this zone.[3]

Three cases are recorded[4] in whom—in addition to fits occurring spontaneously in the usual way—slight cutaneous irritation in a definite area produced an epileptic seizure. In Dr. Jackson's case, a boy aged 8, a flick with a silk handkerchief on the back of the head from behind, so that he received no warning, would result in his immediately falling down in a fit. In 1897, at the age of 19, this boy was under my care, and he presented hemiplegia and unmistakable signs of descending sclerosis and commencing athetosis. It is extremely interesting to notice that all these cases presented important features in common. All were children, two boys and one girl, and in all the fits began early in life. In all the epileptiginous zone was situated in the head, and it was only when the patients were unaware that they were going to be touched that fits were produced. In falling, it was noticed that these patients generally struck their heads with great violence, suggesting that the clonic convulsions began in the neck muscles, pulling down the head in one direction. Finally, there were evidences in all pointing to a gross lesion of the brain as a cause of the fits. The two boys were hemiplegic, and this symptom was increased after the fits. The girl was not hemiplegic, but in her the history of a series of convulsions at the age of 3, followed by two years interval, points in the same direction (see § 593 below).

Treatment. Bromides constitute the sheet anchor in the treatment of idiopathic epilepsy. Personally I prefer the ammonium salt in doses of 20 grains twice daily, gradually increased to 60 grains until the fits cease to occur. The administration should continue for two full years after the cessation of the fits. When thoroughly under the influence of bromide the palate reflex is lost. For nocturnal epilepsy add digitalis to a single dose of bromide. If bromides fail, borax, belladonna, picrotoxin (or a combination of this with bromide, known as Gelinot's dragées), amyl nitrite or liquor trinitrini 3 or 4 times a day may ward off an attack of E. major, but the 2 last aggravate E. minor. Morphia has been successful where other things failed. A regular life aids; and excess of animal food (of which these patients are very fond) and of salt, should be avoided. Possible causes of reflex irritation should be removed. The treatment during an attack consists simply in placing something between the teeth and preventing injury. The *status epilepticus* may be ameliorated by amyl nitrite, chloroform, chloral hydrate by rectum, morphia and ice to the spine.

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[1] *Brain*, 1895.

[2] Med. Soc. Trans.

[3] Gowers, "Diseases of the Nervous System."

[4] James Dunsmure, *Edinburgh Med. Journ.*, Oct., 1874; Hughlings Jackson, *B. M. J.*, 1886, ii., p. 962; Wilfred J. Harris, *Lancet*, ii., 1897, p. 537.

II. **Hysterical convulsions** constitute 18·5 per cent. (Gowers) of cases of convulsions. They form a frequent manifestation of hysteria occurring in my experience in about 30 per cent. of the cases. The convulsions consist of (1) tonic spasm, often amounting to opisthotonos, *alternating* with (2) clonic spasms, often of great violence, lasting longer than true epilepsy, and usually with purposive movements, such as dashing the head against a pillow, and struggling with those restraining her. The patient may then have a succession of fits for a few hours. The severer form described by Charcot, Richer, and others as hystero-epilepsy consists of 4 stages—tonic and clonic spasms, emotional attitudes and delirium. Trance or lethargy may follow, and last for days. The *diagnostic* features of a hysterical from an epileptic fit are—(1) the onset dates from some emotional disturbance, and it is not preceded by a definite aura, though it is frequently preceded by the globus or an epigastric sinking. (2) The convulsions do not follow the regular order of epilepsy: there is more tonic rigidity throughout, and many of the clonic movements have a distinctly purposive character. (3) The consciousness is not excepting in rare cases, lost: the conjunctivæ are sensitive and the patient may be talking nonsense all the time (which never occurs in epilepsy), though she may not afterwards remember who was there. The urine and fæces are very rarely voided. On recovering the patient may burst into tears or laughter.

Hystero-epilepsy as described by Charcot is very rare in England. In all my experience at the Paddington Infirmary I only saw 3 cases, one of which was that of a man aged 29, whose attacks were of great violence, and consisted of 4 stages: (1) a tonic, (2) a clonic, (3) a stage of purposive movements, and (4) a stage of incoherent delirium.[1]

III. **Intracranial Syphilis** is the most frequent cause of convulsive fits occurring for the first time over 30 years of age. Convulsions are, moreover, a very frequent manifestation of cerebral syphilis (Charcot and others). Sometimes the convulsions are due to gumma, meningeal thickening, or a diffuse meningitis, but occasionally no gross changes are found beyond congestion of the cortex. In gummatous cases the convulsions have Jacksonian characters (see below), but in other cases the fits are indistinguishable from idiopathic epilepsy excepting by attendant circumstances—namely (1) the age of the patient; (2) severe headache, which is present in over 77 per cent. of the cases and heralds the attack—continuous, bilateral, markedly worse at night, and getting progressively worse up to the attack. Pains in the limbs may also be present. (3) There is more often mental hebetude between the fits. (4) If the lesion be gummatous there may be scattered lesions of the cranial nerves, and especially optic neuritis, or partial transient palsies or transient aphasia.

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[1] Clin. Soc. Trans., 1889.

IV. **Irritative lesions of the cerebral cortex** are always attended by convulsions. (1) JACKSONIAN EPILEPSY may be caused by a syphilitic gumma or other tumour, or a cicatrix in the brain after operation, injury, or hæmorrhage, *occupying the cortex* in the motor area (Fig. 150). (i.) When a lesion irritates one of the motor areas of the cortex, the clonic spasms are always partial, at any rate at the outset, the convulsions starting in and sometimes limited to the fingers or toes, one forearm or leg, or the face on one side. The point of starting indicates the precise position of the lesion in the cortex. The clonic spasms may remain limited, or may gradually spread to the rest of the body. (ii.) No loss of consciousness, unless the movements become general and very violent. (iii.) The attacks are apt to follow in frequent succession in a series. (iv.) There is no aura; as a matter of fact the twitching of the thumb, or big toe, as the case may be, constitutes a motor aura, and is known as the signal symptom.

(2) HÆMORRHAGE and EMBOLISM of the brain are attended by an epileptiform seizure in about half the cases. They are more frequent in hæmorrhage than in embolism, but this fact has not the value for purposes of diagnosis, which was attributed to it by the older authors. The convulsions under these circumstances usually predominate on one side, are at first attended by tonic spasm, and later by paralysis on the side opposite to the lesion. The age and history also aids in the diagnosis. Mitral stenosis favours the diagnosis of embolism.

(3) Various CHRONIC DEGENERATIONS of the nervous system are sometimes attended by convulsions. The most frequent, perhaps, of these is general paralysis of the insane, especially in the congestive form. They are comparatively rare in disseminated sclerosis.

(4) ACUTE MENINGITIS (at the outset) and HYDROCEPHALUS in children, and PACHYMENINGITIS in the adult are frequently attended by convulsive seizures, but these have not Jacksonian characters. They are said to be relatively infrequent in alcoholic meningitis and cerebral ABSCESS (Gowers).

V. **Uræmic convulsions** are associated with renal disease, especially the chronic interstitial form. Convulsions, especially if associated or alternating with coma, occurring suddenly for the first time in a person over 50 years of age, offer a presumption of uræmia or apoplexy. Uræmia is distinguished from apoplexy by (1) the absence of hemiplegia, and the pupillary reactions (§ 525); (2) an examination of the urine reveals renal disease. Finally, it should be remembered that apoplexy and uræmic convulsions may be present at the same time.

VI. **Puerperal Eclampsia** (syn. puerperal convulsions) is the term applied to convulsions occurring before, during, or after labour, and they only differ from idiopathic epilepsy in the circumstances under which they occur. They are practically always associated with albuminuria, and are in all probability of toxæmic origin. They may be associated throughout with some rise of temperature. Bleeding and the transfusion of normal saline fluid (§ 107) has been warmly advocated. Chloroform and chloral are very useful.

VII. Various **other toxæmic** conditions may be attended by epileptiform convulsions such as—(1) ALCOHOLISM, especially in a chronic alcoholic after a drunken orgie. (2) LEAD POISONING occasionally, when other symptoms of plumbism would be present. (3) Various DRUGS given in overdose or attempted poisoning—strychnine (when the convulsions are mainly tonic) belladonna, strophanthus, stramonium, aconite, veratrium viride, sabadilla, hydrocyanic acid, in all of which the convulsions come on suddenly, other symptoms of the drug are present, and the case terminates one way or the other in 24 hours. (4) TETANUS (in which the spasms are chiefly tonic), and MALARIA in adults, and the onset of the ACUTE SPECIFIC FEVERS in children. (5) Convulsions also occur with DIABETES and with cholæmia in the terminal stages of DISEASES OF THE LIVER such as acute yellow atrophy.

VIII. **Cardio-vascular disorders** give rise to epileptiform seizures in adult and advanced life more frequently than is generally supposed. Kussmaul has shown experimentally[1] that either anæmia or congestion of the brain may produce convulsions, and we know that profuse hæmorrhage in cut throat cases and cases of asphyxia may be so attended. This explains their occurrence in cardiac weakness, especially when associated with vascular disease in the aged; and in congenital heart disease in youth.

Adams-Stokes Disease, which illustrates the same principle, and has been already briefly referred to on p. 129, is a rare disease first described by R. Adams[2] in 1827, characterised by a permanently slow pulse with syncopal and epileptiform attacks. The patients are usually advanced in years, complain of dyspnœa, and have marked bradycardia, the pulse rate ranging from 20 to 40. Any mental excitement is liable to bring on an attack. The breathing becomes stertorous, the face cyanosed, there is dilatation of both pupils, rigidity of the body accompanied by clonic movements of the arms, the pulse beats more and more slowly, and finally ceases, the jaw drops, and for 40 to 80 seconds the patient is to all appearance dead. No pulse is felt in either wrist and on auscultation the cardiac sounds are inaudible. Then a feeble sound is heard followed by a stronger, and a second later the pulse begins beating (one can feel the artery fill), the cyanosis lessens, the pupils contract, and consciousness returns. Many such fits may occur in succession, from six to ten in a single night. There is no albuminuria.

The *etiology* is uncertain. Irritative lesion of the vagus, or disease of the cardiac ganglion has been suggested. Osler considers it has a close connection with angina. Treatment is directed to the bradycardia (§ 60); inhalations of strong ammonia may arrest an attack. Strychnine and bromides are recommended.

IX. **Reflex causes** occasionally produce epileptiform convulsions. In addition to those already mentioned under the etiology of idiopathic epilepsy, it may be remembered that washing out the pleural cavity after aspiration, and severe renal and biliary colic are occasionally followed by convulsions. Intestinal parasites, a tight prepuce, and any chronic peripheral irritation may be mentioned.

§ 593. Convulsions in infancy and **childhood** may be produced by some of the foregoing conditions, but owing to the susceptibility of the nervous system in childhood, other and slighter causes may also produce convulsions. Judging by the frequency with which slight causes are followed by convulsions in children, it seems as though the instability of the cortical motor cells, which constitutes the essential disorder in idiopathic epilepsy, exists as a normal condition in infancy.

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[1] Publications of the New Sydenham Society.

[2] Dublin Hospital Reports, iv., p. 396. See also *The Lancet*, 1885, vol. i., pp. 10, 288, and 447; also June 4, 1904, p. 1569, and Feb. 13, 1904, p. 411.

(1) *Blood poisons.* The acute eruptive fevers in adult life may be ushered in by a general tremor (rigor), but in childhood their advent is frequently marked by generalised convulsions. The advent of infantile paralysis, acute pneumonia and many other acute diseases might also be mentioned. Rickets is often accompanied by convulsions, probably due to gastro-intestinal toxæmia. (2) *Reflex causes.* Constipation, worms, or any other irritative condition of the alimentary canal, teething and the like, are frequently attended by convulsions caused either in a reflex or toxic manner. (3) *Gross lesions of the brain*, such as injuries to the brain at birth, hydrocephalus, tumours, abscess following otitis media and some of the other gross conditions above named. Meningitis should be suspected if there is retraction of the head, peevishness on movement, or squint. Many of the causes of tetany, trismus, and carpo-pedal contractions may also give rise to convulsions. (4) In children under 10 *cerebral hæmorrhage* may occur, with convulsions, usually followed by hemiplegia (§ 518). In children a small cortical hæmorrhage or embolism may occur without a definite or very noticeable hemiplegia, and a few years later the cicatrix may be sufficient to cause recurring fits practically identical with idiopathic epilepsy. When fits have commenced under 10 years of age, careful inquiry should be made of the mother as to whether any hemiplegia accompanied or preceded the first fit. It has been estimated[1] that in about one-half of these cases, the convulsive seizures date from the occurrence of the lesion: but in the other half they do not commence until some time afterwards. In some cases convulsive attacks preceded by an aura and indistinguishable from idiopathic epilepsy may continue at intervals throughout life, but in other cases the clonic spasms are unilateral or Jacksonian in character. See also cases referred to on p. 1053, *ante.*

On the whole, the *prognosis* of infantile convulsions is much more favourable than the same symptom in adults, a statement which follows from the opening considerations. In the *treatment* attention should always be first paid to the intestinal canal which is so easily upset in children, and as a general rule, the administration of castor oil and lime water in equal parts, a teaspoonful every 4 hours, is a good adjunct to other measures of treatment. Hyd. cum. cret., magnesia and soda are also useful. The cause in 9 cases out of 10 will be thus relieved, but careful inquiry should be made for other possibilities. To relieve the convulsions, bromide, preferably of ammonium, in 1 to 3 grain doses is very useful, and it may be given with ½ to 2 grains of chloral. In severe cases chloroform may be administered, and a very little is sufficient to relieve the spasms.

GROUP IV. δ. MUSCULAR ATROPHY.

§ 594. Muscular wasting occurs in two groups of disorders: (1) those due to a lesion of some part of the lower motor neuron (the peripheral nerves or their cells of origin in the anterior horns of the spinal cord), when it is spoken of as atrophic or amyotrophic paralysis, or briefly as amyotrophy; (2) those due to a lesion in the muscle itself, when it is spoken of as myopathy, or idiopathic myopathy.

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[1] Hare, "Practical Diagnosis." London, 1897, p. 482 *et seq.*

In the CLINICAL INVESTIGATION there are several important *fallacies* to be borne in mind. In the first place, the amyotrophies must not be confused with the general wasting due to constitutional debility (Chapter XVI.). (2) They must not be confused with the different forms of upper neuron paralysis which, when of long standing, are sometimes followed by a slight degree of wasting from disuse; or, (3) the wasting consequent on disuse after the application of Sayre's jackets or other apparatus.

The *clinical features* to investigate are—(1) the flaccidity of the paralysis, the absence of the knee-jerks, and the electrical alterations, which are the three features (in addition to atrophy) which distinguish lower neuron paralyses. (2) The distribution of the amyotrophy. (3) The presence of sensory alterations is of value to localise a lesion. (4) The mode of onset of the disease; and (5) the age of the patient, are also important.

Amyotrophies without sensory changes.

Anterior horns of spinal cord.	I. Acute anterior poliomyelitis (rare in adults). Ia. Acute and sub-acute anterior poliomyelitis of adults. II. Progressive muscular atrophy (rare in children). III. {Amyotrophic lateral sclerosis. Bulbar paralysis.	Flaccid atrophic paralysis, with R. D. No sensory changes and no pain after onset.

Amyotrophies with pain and sensory changes.

Nerve trunks.	IV. Plexus paralysis. V. Localised neuritis. VI. Multiple neuritis.	Flaccid atrophic paralysis with pain and sensory changes. R. D. present.

Myopathies.

Muscles only.	VII. Myopathy with loss of volume:— Erb's juvenile myopathy. Infantile myopathy.	Simple muscular weakness with loss of volume, predominating in one or other place. No pain, or sensory changes. No R. D.
	VIII. Pseudo-hypertrophic paralysis.	Same as preceding, but with increase of volume.
	IX. Peroneal type (doubtful whether muscular or neuro-muscular).	May also have slight sensory changes and fibrillary twitchings.

Reflex amyotrophy.

?	X. Arthritic atrophy.	Extensor paralysis.

§ 595. I. Acute anterior poliomyelitis (syns. poliomyelitis, infantile paralysis, atrophic spinal paralysis) may be defined as a disease of infancy, coming on suddenly with feverishness, characterised by rapid wasting and loss of power in one or more groups of muscles, due to inflammation of the anterior cornua mostly located in the lumbar or cervical enlargements. Clinically the malady has considerable resemblance to an acute infection, and epidemics have been observed. Histologically the lesion is a capillary thrombosis not a true inflammation, and it is now coming to be regarded (Marie, Batten, and others) as microbic in origin.

Symptoms. (1) The patient is usually about the age of the first dentition, very rarely older than 14 years. The advent is sudden, and is ushered in by moderate fever, general prostration, and more rarely by convulsions. Pains in the limbs may be complained of at the outset, but do not form a prominent feature. (2) Sometimes the paralysis attacks only one limb, sometimes all four; and it not infrequently takes the form of paraplegia. At first (in the course of 24 to 48 hours) all the muscles of the limb or limbs are affected with flaccid paralysis and rapid wasting, and they are tender to touch or movement. In a few weeks some of the muscles begin to recover, while the others undergo progressive wasting. All the affected muscles show the reaction of degeneration as early as a week after the attack. (3) The superficial and deep reflexes are abolished in the paralysed parts; the knee-jerk remains lost only when the quadriceps is permanently paralysed. (4) There is absolutely no affection of sensation. The bladder and rectum are not usually affected, and there is no tendency to bedsores. (5) In the course of a few months the amyotrophy settles down into a muscle or group of muscles—for instance, in the leg the tibialis anticus or quadriceps (more rarely the hamstrings or glutei), and in the arm the deltoid, brachialis or supinator. These muscles atrophy, degenerate, and finally, a year or so later, undergo contraction, producing various characteristic and familiar deformities (club-foot, etc.). Some hold that the deformities are due to contracture of the atrophied muscles; and some that they are due to neuro-trophic changes in the joints and ligaments; but the unopposed contraction (which is constant) of the unaffected muscles has always appeared to me sufficient, bearing in mind the age-period at which the disease occurs. The affected limb becomes blue and cold, and the bones do not grow so much as on the healthy side. It seems probable from this that the anterior cornual cells control the tone of the involuntary as well as the voluntary muscles of the part.

Diagnosis. The disease is practically confined to early childhood. At the onset the malady is mostly mistaken for a "chill," or the onset of an acute specific fever. In the pseudo-paralysis of *rickets* there is no atrophy. In *meningeal* affections the spinal pain is very marked, and the muscular atrophy is not so pronounced. *Peripheral neuritis* has more pain and does not produce such a rapid or localised amyotrophy. In old-standing cases the history of acute onset differentiates it from *idiopathic myopathy. Syphilitic* pseudo-paralysis due to separation of the cartilage at the end of the diaphysis has crepitation and pain on movement. In *infantile scurvy* there is tenderness and swelling of the affected limbs.

Prognosis. The disease is not fatal in itself, but as a rule complete recovery is rare. Within a few weeks or months after the acute disturbance has subsided, the muscles may usually be grouped into three groups, (a) those which will recover of themselves, and in these the electrical changes are but little altered. (b) Those which may recover under treatment. In these the galvanic changes are typical, *i.e.*, increased with A.C.C. > K.C.C., and the faradic reaction is not quite lost. (c) Those which probably will not recover under any treatment, and in these faradism gives no response whatever. Death may occur from pneumonia or other intercurrent diseases.

Etiology. The disease occurs chiefly in children under ten, usually about the age of two or three years. It only occasionally affects adults. Chill has been supposed to cause it : and it has appeared during convalescence from acute diseases, and during pregnancy. It is more frequent in summer, and has occurred in epidemics. Its clinical course resembles an acute microbic disease.

Treatment. At first rest in bed, salines, and quinine or salicylates are all that can be ordered, with anodynes if there be much restlessness. At the end of a few weeks treatment should be begun for the atrophied muscles. For these the galvano-faradic current, or the galvanic current alone, followed by massage and shampooing form undoubtedly the most efficacious treatment.

Electricity or massage alone is not nearly so valuable. It is generally held that no treatment is of any use after a few years have elapsed, but I have obtained considerable benefit in patients even 20 and 25 years later. The treatment of the deformities which result is surgical.

Ia. **Acute and sub-acute poliomyelitis** in adults (syn. atrophic spinal paralysis of adults) is almost the counterpart of infantile paralysis, occurring between 25 and 30 years old. It commences with fever and pains in the back, which may last a week or two, accompanied or followed by paralysis, frequently of both arms and legs. In the sub-acute form one group of muscles after another may become paralysed till the whole body is affected. The disease is frequently mistaken for multiple neuritis (§ 556), in which, however, pain in the limbs is a prominent feature. Time clinches the diagnosis, for whereas recovery is nearly always perfect in multiple neuritis, groups of muscles remain permanently paralysed when the disease is acute or sub-acute anterior poliomyelitis. The latter is certainly very rare, and most cases so diagnosed have probably been multiple neuritis.

§ 596. II. The chronic form of anterior poliomyelitis in adults is now considered identical with **progressive muscular atrophy.** It is not a common disease, but is of more frequent occurrence than the preceding. It is recognised by (1) a slowly progressive atrophy of the muscles, starting in the thenar and hypothenar muscles, usually of both hands, and spreading thence to the muscles of the arms and scapulæ. (2) The patient is generally an adult of 30 or 40 years of age, or more, very rarely younger. (3) Partial reaction of degeneration is present; there is progressive loss of faradic and galvanic contractility precisely as the muscle substance wastes (Ferrier). (4) There are no sensory symptoms. Owing to the early wasting of the interossei there are hollows between the metacarpals, and from the unopposed action of the extensors the first phalanges are over-extended, the two distal phalanges flexed—the typical 'claw hand," or "main en griffe." The muscles show the fibrillary twitchings which used wrongly to be regarded as pathognomonic. Instead of the hand muscles, the deltoid may be the first to atrophy, or the extensors of the forearm. Later the supra- and infra-spinatus, the lower two-thirds of the trapezius, and the trunk muscles are involved. The legs are spared till very late; the knee-jerk goes when the least sign of atrophy of the thigh muscles is present.

The *diagnosis* is not usually difficult on account of the peculiar limitation to the hand in its earlier stages, and its bilateral distribution. In local atrophies, due to single nerve paralyses (§ 564), a history of a cause is usually present. In other forms of *neuritis* the paralysis follows a certain nerve distribution, and sensory changes are usually present. The peroneal type of muscular atrophy (§ 597) begins usually in childhood, in the peronei, or extensor muscles of the foot; some years later the hands and arms atrophy, and the claw hand may be produced: the condition is possibly a neuritic affection. In *cervical pachymeningitis or tumour* there are paroxysmal pains and stiffness, or twitchings of the muscles. In *amyotrophic lateral sclerosis* the muscles are rigid and the tendon reflexes increased. *Chronic disseminated myelitis* has symptoms of irregular damage to other parts of the cord. The *idiopathic myopathies* may be difficult to differentiate, but as a rule they commence at an earlier age and other members of the family are affected. *Syringomyelia* produces amyotrophy of the arm or arms, but is attended by sensory symptoms and lateral sclerosis.

Prognosis. The disease progresses very slowly. It may start in one arm first, and the other may not be affected for a year or longer. On the other hand, after a fairly rapid progress at the commencement, it may become stationary. The hand may be useless in 6 months, the arms in 2 years. The prospect of arrest is better in symmetrical cases; but is always bad in old age. Complications arise when the chest muscles are affected. The disease may spread to the medulla, when the symptoms of bulbar paralysis may ensue. On the other hand, the speech may be impaired for years without further symptom of disease in the medulla.

Treatment. The course of the disease may be considerably delayed by appropriate treatment by galvanism and massage, with strychnine, which is more efficacious when given hypodermically, in the form of the nitrate, gr. $\frac{1}{30}$—$\frac{1}{20}$, once daily for months. Cod-liver oil and other tonics are also given.

III. Certain other mixed or overflow *lesions of the spinal cord* are attended by amyotrophy.

The amyotrophic lateral sclerosis of Charcot is a very rare condition which commences with atrophy of one of the upper extremities, attended by increased reflexes. Following this, after an interval of a few months, are the signs of descending sclerosis in the leg of the same side. The lesion gradually spreads to the pons, and the patient rarely lives more than two or three years. No known remedy seems to have any effect upon its course.

Bulbar paralysis, or glosso-labio-laryngeal palsy, consists of atrophic paralysis of the muscles supplied by several of the cranial nerves (§ 619).

Amyotrophy with pain and sensory changes.

Amyotrophy attended by *pain, numbness*, or other *sensory changes* is due to disease or injury of the *nerves, nerve trunks*, or *plexuses*, all of which it will be remembered are parts of the lower motor neuron.

IV. **Plexus paralysis** due to lesions involving the nerve plexuses (§ 565).

V. **Single nerve paralysis,** *e.g.*, wrist-drop due to musculo-spiral paralysis consequent on injury or lead poisoning (§ 564).

VI. **Multiple neuritis,** usually the result of some toxaemia such as alcohol (§ 556).

Myopathies.

§ 597. **Myopathy** or idiopathic muscular atrophy (syns. primitive myopathy, muscular dystrophy) is a somewhat rare condition full of obscurity. It may be defined as muscular wasting starting in certain groups of muscles, and tending to become generalised, due it is believed to changes in the muscular substance, of hereditary origin. It was first studied and carefully described by Duchenne de Boulogne in 1850. Since then it has been studied by many different observers, whose names have been applied to the particular sub-variety which has come under their notice. These varieties however only differ in (1) the particular age at which the symptoms were first observed, (2) the particular group of muscles first involved, and, of course, (3) following Duchenne's original description.[1]

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[1] The two chief types described by Duchenne were the Hereditary Infantile Muscular Atrophy and Pseudo-hypertrophic Paralysis—Sur l'Eletrisation Localisée (Sydenham Society's publication).

whether the muscles are diminished or increased in volume, depending on the amount of interstitial fibrous tissue present in the muscles.

The features *common to all varieties* of this curious disorder are—(i.) the presence of a hereditary taint, and the fact that several of the same family may be affected. (ii.) The disease dates from very early in life, though it may not be noticed till adolescence, or even later. (iii.) It runs a very slow course, lasting sometimes throughout life, though the patient generally dies of some intercurrent malady about 30 years of age, or earlier. (iv.) The muscular wasting, wherever it may begin, becomes widespread, and finally involves all the muscles. (v.) There is said to be no reaction of degeneration, but cases have been recorded by the author[1] and others, in which electrical changes have occurred. As a rule the electric irritability is diminished equally to both currents in proportion to the degree of wasting present. (vi.) There are no sensory changes.

Idiopathic myopathy is met with clinically in two principal types, based on loss or increase of volume in the muscles. In both there is an atrophy and final disappearance of the muscle fibres, but in the latter there is a great increase of the interstitial fibrous tissue.

VII. **Myopathy with loss of volume** and without initial hypertrophy of the muscles (the hereditary infantile amyotrophy of Duchenne) has been grouped into two types.

α. Cases beginning in youth or adolescence (Erb's Juvenile Myopathy).

β. Cases beginning in childhood and infancy. (i.) Form without involvement of the face. (ii.) Form with involvement of the face (the facio-scapulo-humeral or Landouzy-Déjérine type. (iii.) Form beginning in the legs (rare). In most idiopathic myopathies the muscular wasting *begins* and predominates in the biceps, triceps, and supinator longus, and the shoulder muscles, the latissimus dorsi, teres major and lower two-thirds of the pectoralis major. The hands are rarely involved. In the Landouzy-Déjérine type the face muscles are first attacked; the naso-labial fold is lost, the lips are apart, the lower lip projecting, and a peculiar dull expression is present. In a good many cases first noticed in adolescence or later there is a history of the patient being unable to close the eyelids properly from earliest childhood.

VIII. **Pseudo-hypertrophic paralysis,** or **Duchenne's paralysis** (Fig. 5, p. 30), is the only form of idiopathic myopathy in which there is an increased volume in the muscles, and this is due to an increase in the interstitial tissue. The clinical features by which it may be recognised are (i.) the patient is always a child, the disease having been first noticed between the ages of 4 and 14, and he is generally brought to us for "weakness of the legs." (ii.) On account of the disease predominating in the legs, the walk is a waddle, and very characteristic, and when the patient lies down, he is unable to get up without clambering up by placing his hands on his own knees. (iii.) There is a notable increase in the volume of some of the muscles, especially of the calves and buttocks, which gives the child the appearance of an infant Hercules. The muscles in pseudo-hypertrophic paralysis which are increased in volume are the calf muscles, the glutei, deltoid, supra- and infraspinati. Muscles which are apt to be diminished are the lower two-thirds of the pectoralis major, the latissimus dorsi, and the teres major. The weakness of the muscles of the shoulder girdle leads to a very characteristic symptom, namely, when one endeavours to lift the child under the arms the shoulders slip up to the ears. The hand muscles escape. Deformities such as lordosis and talipes equinus occur from the weakness and contracture of the respective muscles of the spine and legs.

As in the myopathy with diminished volume, heredity is a potent cause, and one may find in members of the same family examples of this disease and the other forms of primitive myopathy, showing that the cause, whatever it may be, is a developmental one, and that the diseases are identical. This is further proved by the occurrence of both wasting and increase of volume even in the same patient.

Diagnosis of primitive myopathy. The gradual onset and family history distinguish this disease from acute anterior poliomyelitis, which is the usual cause of muscular atrophy in children. Pseudo-hypertrophic paralysis is not difficult to diagnose by reason of the enlargement of the calves associated with weakness of the shoulder muscles. In neuritic muscular atrophy (*e.g.*, lead palsy and multiple neuritis) paralysis exceeds the atrophy and sensory symptoms are usually present. Progressive muscular atrophy begins in the hands and develops at a later age, and there are fibrillary twitchings. Neuro-muscular atrophy of the peroneal type is described below.

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[1] *Brain*, vol. xviii., p. 605.

Prognosis. All cases of muscular dystrophy have a progressive course, though very slow, lasting from 10 to 50 years. Death occurs by intercurrent maladies. In pseudo-hypertrophic paralysis the prognosis is grave, the child often dying before adult life; Gowers says that they rarely live for 7 years after the power of standing is lost. Sometimes, however, the disease remains stationary for many years.

Etiology. The disease is hereditary, but symptoms are most frequently noticed before puberty; males are more often attacked than females. It is transmitted generally through the mother, she being healthy. These muscular dystrophies are closely associated on the one hand with progressive muscular atrophy, due to lesion in the anterior horns of the cord, and with the neuro-muscular or peroneal atrophy which is probably due to changes in the nerves.

Treatment. The progressive wasting appears to be retarded when carefully selected gymnastic exercises are given, and electricity and massage may aid. In the pseudo-hypertrophic form it is important to retain the power of walking as long as possible.

IX. **Progressive neuro-muscular atrophy** (syn. peroneal type of muscular atrophy) is associated with the names of J. M. Charcot, Pierre Marie, and Howard Tooth, who have described it. It forms a connecting link between progressive muscular atrophy and the muscular dystrophies above described. Like the dystrophies, it is hereditary and commences in childhood. It is of gradual onset, usually attacking first the peroneal muscles, leading to club-foot (pes equino-varus), but it may begin in the hands. The intrinsic muscles of the hand are affected as a rule several years later than the legs. There are fibrillary twitchings, slight sensory changes, and diminished excitability to electricity with reaction of degeneration. Gradually the wasting extends all over the body. When a case is first seen it may be difficult to *diagnose* from an old-standing case of acute anterior poliomyelitis, but the latter is not hereditary, and has no sensory symptoms. As a cause of acquired club-foot this disease should be remembered, and "claw hand" in childhood is practically always due to this disease.

X. **Arthritic Amyotrophy.** It was, I believe, Charcot[1] who first pointed out that joint lesions are sometimes followed by muscular wasting, quite independently of the disuse which may attend the injury or disease of the joint, since it may supervene too rapidly to be explained in that way. It may follow a contusion or joint trouble which was so slight as to be overlooked. The atrophy in such cases has three clinical features—(i.) it is limited to the muscles which move the affected joint; (ii.) it predominates in the extensors; and (iii.) there are no qualitative electrical changes, only a quantitative diminution. When the arthritic trouble is cured, massage and electricity will speedily restore the amyotrophy.

GROUP V. PAIN AND SENSORY SYMPTOMS.

Pain.

Pain is a symptom which has been referred to many times in this work, and the importance of investigating its position, character, degree and constancy has already been insisted on. Pain is present in many nervous disorders, but it is almost the only symptom in Neuralgia and Migraine, two disorders which will now be considered.

§ **598. Neuralgia** may be defined as a paroxysmal pain referred to the seat of some sensory nerve or its branches, in the absence of any obvious local organic affection.

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[1] "Leçons Cliniques sur les Maladies du Système Nerveux," tome iii.

The pain of a true neuralgia is usually described as sharp, shooting, or knife-like, with *paroxysmal exacerbations*, and between the exacerbations there is a dull aching pain. It is usually associated with localised tender spots, especially at certain points called the "points of Valleix," by whom they were first studied. *Destructive* lesions of a sensory or mixed nerve cause mainly anæsthesia. *Irritative* lesions of the spinal ganglia (or their analogue the Gasserian ganglion), or of the sensory fibres on either side of these, cause pain and may be attended by vascular dilatation, redness, œdema, and various trophic changes of the area of skin in which the sensory fibrils terminate. Herpetic vesicles are very prone to appear[1]; and, in long-standing cases, atrophy of the skin (glossy skin) and subcutaneous tissue ensue.

Clinical investigation and *causes of neuralgia.* The causes differ somewhat with the seat of the neuralgia, but certain general causes may be mentioned in the order in which the examination of the case should proceed, viz., (a) local, (b) reflex, and (c) constitutional causes. (a) A careful *local* examination should always be made of the nerve trunk chiefly involved, *i.e.*, in the position where the pain started, to see if it be the seat of tenderness, or other signs of irritation. The parts beneath and around the nerve should also be examined to see whether it be pressed upon by a tumour or disease of the bone through which it passes, or any other lesion; for instance, what appears to be an intercostal neuralgia may be due to a small patch of dry pleurisy, or the pressure of a swollen gland, or rheumatic nodule. (b) In many cases of true neuralgia, some *reflex cause*, sometimes of a very trivial nature, is in operation, and the discovery of this depends very much on the clinical acumen of the physician. This is especially true of trifacial neuralgia, where a source of irritation in the teeth or some other part of the body is so frequently overlooked. The teeth may, to all appearances, be perfectly sound, and yet, as I have frequently found, an inflamed or irritated pulp may be revealed by the tenderness of some tooth when tested by tapping or the application of hot and cold liquids alternately. (c) *Constitutional* and general conditions act mainly as predisposing factors. Neuralgia is essentially a disease of adult life; it is rare in children, and not common at the other extreme of life. It is said to be more common in the female sex, and a neurotic family history is often obtainable. Neurasthenic, nervous and hysterical subjects are certainly more sensitive to pain, and more liable to be attacked by neuralgia. Anæmia is often in operation, and over-fatigue of mind or body, anxiety, over-lactation, unhygienic surroundings, or any other debilitating influence, may predispose to or determine an attack. Convalescence from an acute illness, tuberculosis, and various other cachectic states afford predisposition to attack. Neuralgias are frequently associated with gout, rheumatism, and ague. Many other toxic influences may give rise to neuralgia, such as alcoholism, plumbism, diabetes, and the other conditions mentioned under peripheral neuritis (§ 556) may be heralded by neuralgic pains. Syphilis may produce neuralgia in two ways; the nerve may be pressed upon by syphilitic disease of the periosteum, theca, or bone through which it passes; or the nerve may itself be the seat of a neuritis of syphilitic origin. The former is the more frequent. Among the general exciting causes, exposure to cold and chill is by no means infrequent.

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[1] I have elsewhere shown that herpetic vesicles are a definite indication, not of a destructive, but an *irritative* lesion. *Clinical Journal,* Sept. 7, 1898.

General remarks on the *treatment of neuralgia*. In all cases the cause should be carefully sought for and if possible removed. Treatment should be directed to any dyscrasia present, such as gout, rheumatism, ague, anæmia, the latter especially offers a predisposition to the development of the different neuralgiæ. For the rest, the treatment resolves itself into—tonics, hygienic measures, palliative measures, and radical measures. Among *tonics* quinine (in large doses) and arsenic take a leading place: iron, phosphorus, cod-liver oil and strychnine are also useful. Nervous excitants, such as strychnine, should be avoided when the nervous system is exhausted, and nervous sedatives, such as bromides, administered when the nervous system is irritable. The *nutrition* should be attended to, cod-liver oil, and two or three pints of milk a day being given in addition to other food. Alcohol, as a rule, should be avoided, though it may relieve temporarily. Over-feeding, combined with perfect rest, the liver being attended to, often succeeds where other measures have failed. The patient should be kept in bed, and 11 to 16 hours of sleep per diem should be procured by large doses of bromide. Warm baths are certainly of great value, and I have sometimes been able to cure painful affections that have resisted all other means by Turkish baths, regularly and freely administered. The *internal palliative remedies* are numerous. Among the modern remedies, phenacetin, 5 to 10 grs., antipyrin (combined preferably with caffein citrate), acetanilid, grs. iij. to viij., and chloralamide are useful. It will be observed that many of the analgesic drugs are antipyretic as well, a point of interest when we remember that fever is accompanied by generalised pains; the two facts together support the idea that blood conditions alone are capable of producing pain. Tincture of gelsemium ♏ 10 to 20 every 2 hours certainly allays hypersensitiveness of the nerves, and I have found this combined with 20♏ of tinct. cannab. indica of use in migraine, an attack of which may be sometimes thus aborted. They are also useful in other neuralgias. A favourite palliative prescription of my own is tinct. gelsem. ♏ x., antipyrin, gr. v., am. brom. gr. x., aq. chlorof. ℥ss. every 3 hours till relieved. Croton chloral, paraldehyde, and drugs of that class may also be useful, but their effect on the heart should be watched. Bromides, among which bromide of ammonium I believe to be the best, given regularly, combined with the "rest cure" (*vide supra*), are especially useful when uterine conditions are suspected. Chloride of ammonium in large doses is sometimes useful especially for ovarian neuralgia. In the neuralgia of anæmia, inhalation of amyl nitrite often affords relief. I have often found that glonoin affords relief in neuralgiæ of various kinds; and both these last named remedies are invaluable for angina and pains referable to a labouring heart. Finally we have in morphia a prince of remedies, either hypodermically or internally, but it should never be administered except by the medical man himself, and then in full view of the possibility of the development of a habit. Minute doses of morphia hypodermically may possibly have a curative as well as a palliative action on some neuralgias depending on neuritis.

Chief among *external palliatives* is warmth, whether by hot water, a muff-warmer, roasted cotton wool, or poultices. Counter irritation (*e.g.*, mustard leaf behind the ear), emplastrum belladonna, chloroformum belladonnæ, are useful; and menthol and peppermint rubbed in, veratrin or aconite ointment, or oleate of aconitine painted on a *limited* area are all worth trying. Paquelin's cautery dotted over the course of the pain will sometimes produce marvellous results. Electricity may be employed for neuralgia in two ways. (1) A strong current (faradic or galvanic) causing pain will occasionally remove neuralgia at once. It acts as a counter-irritant and is suitable only for slight idiopathic cases and recent cases of hysterical neuralgiæ. (2) Weak faradism, galvanism (2 to 5 m.a.), just enough to be felt. Place the positive pole on the seat of pain, and the negative pole anywhere; avoid sudden variations in strength of current. Faradism used as a sedative must be extremely weak, and with very rapid interruptions. Employ large rheophores well wetted and gently *slid on* to the desired spot. The pain increases at first but diminishes after a few minutes. The action is analogous to and equal to the mechanical percussion (Gowers). Local application of chloroform by cataphoresis[1] is said to be very valuable.

Radical or operative measures may sometimes be necessary. Acupuncture, nerve stretching, or neurectomy have all been tried with more or (mostly) with less success. The removal of a portion of the nerve certainly relieves for a time, but if the nerve joins again, the pain generally returns, sometimes worse than before, and the risk of trophic lesions is great.

Trifacial neuralgia (trigeminal neuralgia, neuralgia of the fifth nerve, tic douloureux, prosopalgia, "face-ache"). Of all the sensory nerves, the fifth is the most frequent seat of neuralgia, possibly because of its exposed position and tortuous course through bony passages. The sensory branches of the fifth nerve are distributed to the skin of one entire half of the face, the mucous membrane of the mouth, nasal cavities, conjunctiva, and the frontal sinuses: its other functions are dealt with elsewhere (§ 608). The pain may involve any or all of the sensory branches in varying proportions, and may radiate in different directions, and there is usually a considerable degree of tenderness, many tender spots, sometimes redness and œdema. In cases of a purely reflex nature this is all. But irritative lesions of the Gasserian ganglion, or parts of the nerve in front of this, are indicated also by various vaso-motor and trophic troubles. In cases of moderate severity, vascularity and œdema of the face, watering of the eye, sweating, and sometimes muscular twitchings and flashes of light may be observed; but acute or severe irritative lesions of the fifth result in herpes of the face or scalp, sloughing of the cornea, pan-ophthalmitis, and even swelling and ulceration of the gums. In cases running a more chronic course, atrophy of the skin (glossy skin) and subcutaneous tissue of the face, and loosening of the teeth may be observed.

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[1] This is a method supposed to cause the applications to penetrate the skin, and to have a selective action on the nerve fibres. The method is to soak the sponge of the positive pole in, say, a mixture of chloroform and alcohol, equal parts, apply it over the painful spot, and place the negative pole over the nucha. The current should be just as strong as the patient can bear comfortably, and may with advantage be reversed every few minutes.

The *causes* (p. 1065) may be local, reflex, or constitutional. (a) If any of the irritative signs just mentioned be present, we must seek carefully for some *local* irritative or inflammatory lesion affecting the nerve trunk or Gasserian ganglion. That this nerve may be the seat of definite inflammatory lesions is certain from the fact that similar signs to these appear after an irritative lesion of a nerve-trunk artificially or experimentally produced. Such inflammatory lesions may be due to deeply-seated bone disease, *e.g.*, from syphilis or tubercle. (b) The cause of trifacial neuralgia of moderate severity is however more often some *reflex* condition, especially that dependent on affections of the teeth. Decay, the presence of stumps, periosteal disease, or affections of the gums, or an inflamed pulp, should all be suspected. Sometimes these affections are obvious, but the last-named (disease of the pulp) is not always so, and may readily be overlooked for a long period of time, as, for instance, when a late wisdom tooth presses on the second molar from below.[1] The best sign of pulp irritation is hypersensitiveness to heat and cold in the mouth alternately. Distant organs may also be a cause in a reflex manner, and these should always be carefully examined in unexplained cases. A good many years ago, Anstey recorded a case of neuralgia of the fifth produced by injury to the ulnar, and another case due to injury to the occipital nerve. (c) Any of the *constitutional* causes previously mentioned may play their part. Ague is a frequent causal agent in the tropics, where it has earned for the neuralgia of the supraorbital branch the term "brow-ague."

EPILEPTIFORM NEURALGIA (Trousseau) is a violent and intractable form of neuralgia of the fifth, attacking people usually beyond middle life, and consisting of a succession of attacks of acute darting pain. Its ætiology does not materially differ from that of trifacial neuralgia.

The *treatment* of neuralgia is given on p. 1066.

§ **598a. Sciatica** is pain in the course of the sciatic nerve. It is difficult to demarcate sharply between neuralgia and neuritis, and every stage between these occurs in sciatica. Pressure and movement increase the pain, and therefore walking and sitting are difficult. The nerve trunk is tender, and painful points can often be elicited, *e.g.*, near the posterior iliac spine, midway behind the great trochanter and the tuber ischii, and below the head of the fibula. The sciatic phenomenon aids the diagnosis; it consists of flexing the leg upon the hip-joint when the patient is in the recumbent posture; owing to the stretching of the sciatic nerve this process causes pain, which disappears on flexing the leg on the thigh. Symptoms occasionally noticed in chronic cases are fibrillary tremor and slight atrophy. When anæsthesia, atrophy, and partial R. D. are found, the case is one of neuritis, and pressure within the pelvis should be suspected; and if on the left side carcinoma of the sigmoid flexure. Hip-joint disease does not cause pain limited to the sciatic nerve. In spinal disease the pain is usually bilateral.

Prognosis. Sciatica may recover in a few weeks, or it may last for years, especially in the aged. Much depends on the early treatment. It is apt to recur. Complications such as herpes and other cutaneous eruptions are rare. Occasionally the disease ascends the nerve to the cord, with corresponding symptoms.

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[1] "Tomes's Dental Surgery," 4th edition, p. 539.

Etiology. Sciatica affects men more than women in the proportion of 5 to 1. It occurs mostly between 30 and 50, and is unknown under 15. Rheumatism and gout attacking the sheath of the nerve are the chief predisposing causes: anæmic, neurotic people of a rheumatic diathesis also suffer. Exposure to cold is the usual exciting cause. Tumours, especially cancer of the rectum or sigmoid flexure, extension from hip disease, peri- or parametritis, and a loaded rectum, are the chief local causes of sciatica by pressure within the pelvis.

Treatment. In the acute stage rest is all important, and particularly the avoidance of movements such as bending the thigh which stretch the sciatic nerve. Heat is the next most important palliative agent. Mustard plasters and blisters may perhaps abort the attack (see also treatment of neuralgia). In the later stages and for chronic cases galvanism, nerve-stretching, counter-irritants, acupuncture, with or without cocaine or morphia injections, may be tried. The nerve may be stretched daily in chronic cases by forcible bending of the thigh on the abdomen, the foot being flexed all the time. Any rheumatic or other diathesis present must be treated, and remedies which I have frequently employed with great benefit are guaiacum, sulphur, or chian turpentine. See also neuralgia (*supra*).

In **brachial neuritis** (inflammation of the nerves of the arm), the main symptom is severe neuralgic pain shooting down the nerves of the arm. There may also be paresis of a greater or less degree, muscular wasting and the other symptoms mentioned under neuritis (§ 564, single nerve). It is met with in an acute and more or less chronic form; but in any case it is a severe and often intractable condition. Only one arm is usually affected, and the condition may have to be diagnosed from an occupation neurosis by the circumstance under which it occurs.

Any of the *causes* of neuritis may be in operation, but particularly the toxic or constitutional conditions.

I have met with a certain number of cases of brachial neuritis in which the most careful investigation has failed to discover other determining cause than EXPOSURE TO COLD, though some of the patients have been rheumatic or gouty subjects (as in the case mentioned below).

Here is an example of brachial neuritis: A medical man, æt. 55, whose family and previous histories were healthy and free from syphilis. He believed himself to be rather gouty, and was exposed at a theatre to a considerable draught, which impinged upon his left shoulder. Two days later his shoulder joint became acutely painful, but he was unable to discover any physical signs in this position. The pain during the next two or three days became extremely severe, extended down the left arm, and was rapidly followed by a flaccid atrophic paralysis of all the muscles of the upper extremity, though most marked in the ulnar region. In the course of ten days this paralysis had become complete, so that he could hardly move a finger, and the pain, which was of a darting, paroxysmal character, became extremely severe. There were numerous hyperæsthetic areas, and he had tingling and other subjective sensations in the forearm. The fingers, however, were anæsthetic, especially the third and fourth. The most thorough investigation by several physicians of eminence failed to discover any local cause or any physical signs in the joints or other structures of the upper extremity. In the course of time the pain somewhat abated, but the atrophy amounted in ten weeks to a difference of $1\frac{1}{4}$ inch in the upper arm, and $1\frac{3}{4}$ inch in the forearm. The limb was always too tender to test the electrical reactions. At length he derived considerable relief from the pain by Paquelin's thermo-cautery, and with the aid of this and the constant current he slowly regained the use of the arm in the course of 6 or 8 months. Several observers watched this case, but no cause beyond the exposure above mentioned could ever be discovered. He was a "gouty" subject in the sense of having previously had 1 or 2 slight attacks of acute gout

In regard to *treatment* of brachial neuritis, there are two means which generally effect considerable benefit in cases of brachial and other varieties of neuritis due to toxic causes.

(1) The application of Paquelin's thermo-cautery in a white-hot condition rapidly dotted down the painful limb. If the point be made only red, it sticks to the skin and causes great pain; but if white-hot it really causes very little inconvenience, and gives so much relief in these painful cases that the patient asks for more.

(2) The application of galvanism may be of great value to relieve the pain and improve the nutrition of the muscles. See also neuralgia (*supra*).

Neuralgia may affect any of the **other nerves** (sensory or mixed) in the body besides the three types specially referred to above. The chief causes of pain in the various situations will now be briefly mentioned. Head's sensory areas (§ 551) may be consulted in such cases.

Pains in the limbs have been discussed in § 423.

Intercostal neuralgia of an inveterate kind frequently precedes, accompanies or succeeds zoster (shingles), especially in old people. Sometimes it is due to pressure of a tumour on the *spinal nerves*, meningeal thickening, or vertebral disease. If the pain be persistent in a male over 30 years of age, *aneurysm of the descending aorta* may be suspected. The pain may be on one or both sides, and often shoots down the left arm. As in other neuralgiæ, it may be due to a *chill*, or any of the other causes mentioned above.

Neuralgia in the chest is a common accompaniment of affections of the *pleura* and *pericardium*, but is rare in those of the lungs and heart. However, in cases of cardiac valvular disease and disease of the aorta, the pain may occasionally be of a very aggravated character, and may be attended by a sense of suffocation and impending death (angina pectoris).

Mammary and infra-mammary neuralgia are generally the result of *hysteria*, but the organ should be carefully examined for *adenoma* in the young, *carcinoma* in the aged. The word "agony" used by a female patient in connection with the pain is sometimes an aid to diagnosis. Pain in the breast is sometimes reflex from uterine disorder, functional or organic.

Gastric neuralgia is somewhat rare. It is an intermittent pain in the region of the stomach, attended sometimes by nausea and even vomiting, relieved rather than aggravated by food or pressure, unattended by loss of flesh or strength, the digestion being good in the intervals. Gastric pain is one of the four classical symptoms of *Addison's disease*, the other three being progressive emaciation, vomiting, and pigmentation. *Tabes dorsalis* is attended by attacks of severe pain in the stomach (gastric crises). Hydrocyanic acid is worth trying in all intestinal neuralgiæ.

Abdominal pains are described in Chapter IX.

Neuralgic pain in the neck is suggestive of stiff-neck ("rheumatic"), spinal caries or other local affection. Pain in the occiput and back of the neck is frequent in gastric and hepatic disorders. Occipito-cervical neuralgia is a somewhat rare form of idiopathic neuralgia probably of reflex origin.

Spinal neuralgia may arise from *spinal caries*, or some other disease of the spine or theca pressing on the posterior roots. It is generally increased by exertion, and there is a characteristic history. *Meningeal affections* give rise to paroxysms of pain in the region of the spinal nerve roots, shooting round to the front on both sides, or down the limbs, accompanied by great tenderness on movement and perhaps anæsthesia.

Neuralgia of the dorsal spine is a very frequent and troublesome symptom in *neurasthenia* and *hysteria*, in which cases more tenderness may be produced by light touches than by deep pressure, and movement may not aggravate it. Similar to the foregoing is the pain in the spine which forms one of the most intractable results of *railway accidents* (railway spine). It is usually accompanied by tenderness, increased by sitting, or by the vibration of a train. In *dyspepsia or gastric ulcer*, pain is often referred to a tender spot in the mid-dorsal region. A constant grinding or boring pain in the dorsal region, shooting perhaps round to the side and front in a male adult, is always suspicious of *aneurysm*. In the young and delicate, pain in the back may be due to *laxity of ligaments*; under these circumstances it only comes on after sitting or standing upright, and leads to stooping; it is best treated by carefully regulated gymnastic exercises.

Lumbar neuralgia may herald variola, erysipelas, or influenza, or arise reflexly from uterine, renal and other visceral diseases. Lumbago is described elsewhere (§ 442).

§ 599. **Migraine** (syns. megrim, sick headache, nervous headache, paroxysmal hemicrania) may be defined as a headache having a paroxysmal character, and running a more or less definite course, occurring at intervals over a long period of time, generally ushered in by malaise, and frequently associated with and relieved by nausea and vomiting.

In the *symptoms* there are usually two stages. The first stage is attended by some transitory disorder of sensation, such, for instance, as bright spots or dark figures or lines before the eyes (scintillating scotoma). In some individuals other sensations are complained of, such as a feeling of chilliness or of cold feet, or of mental depression, with a dread of impending evil, or restlessness, or pins and needles in the limbs. This stage may last from 5 to 30 minutes or longer, and is then succeeded by the 2nd stage, that of headache, which is usually very severe, and lasts from a few hours to 2 or 3 days. It generally commences in one spot, and gradually involves one half or the whole of the head. It is usually terminated by a feeling of nausea and actual vomiting, after which the headache passes away. It is distinguished from other varieties of headache by its paroxysmal character, periodic recurrence, its definite march, and by its usually being preceded by sensory disturbances and followed by vomiting.

Causes. Megrim is an eminently hereditary disorder. It is like most other paroxysmal neuroses more frequent in the female, affects early and middle life chiefly, especially women at the climacteric. Attacks are certainly predisposed to by any general want of tone in the system ; and determined by anything of a depressing or exhausting nature, physical or mental, such as grief, anxiety, or bodily fatigue, improper food, or impure air.

Prognosis. Megrim usually starts soon after puberty, and the attacks recur until about 50 years of age, when they gradually become less frequent. It is never fatal, but is very often of such severity as to completely prevent the person following her avocation during the 2 or 3 days of an attack.

Treatment. (1) For the attacks. If slight, a cup of strong coffee, or a walk, or drive, or cheerful companionship, will help to stave it off. But if severe, the patient must be kept in bed, and perfectly quiet, in a darkened room, with nothing but iced milk to drink. Bromides, grs. x. every hour with a little sal volatile may be combined with phenacetin, antipyrine, and many of the other drugs mentioned under neuralgia. In some cases, chloride of ammonium, in doses of grs. xv., or guarana powder relieves. If these means fail, any of the following may be tried : caffein, cannabis Indica, croton chloral, gelsemium, and amyl nitrite. The latter inhaled often gives wonderful relief, and strongly supports the theory of vaso-motor origin. That it fails in some cases is readily understood, because the poison, whatever it may be, is still circulating in the blood. (2) Between the attacks, the mode of life should be regulated so as to avoid any known exciting condition. And as regards food, there are two opposite modes of treatment. One is to diminish the diet as much as possible, and especially in its nitrogenous constituents ; the other, with which I have met with more success, is to give a liberal dietary, with copious libations of milk, in addition to the ordinary diet, and two glasses of old Burgundy with each of the two chief meals. Salicylate of soda, in view of Haig's researches, which certainly have much to support them, is a valuable remedy, and should be taken regularly. Morning and evening glasses of hot water to purify the blood, combined with an occasional purge are useful. Tonics, as a rule, are of very little use (see also neuralgia).

Cutaneous Sensation.

§ **600. Disorders of cutaneous sensation** are rare as substantive affections apart from motor defects, excepting in hysterical cases. Moreover, when motion and sensation are simultaneously affected, as after division of a mixed nerve, the loss of sensation is less and is recovered from much more quickly than the loss of motion. Symptoms referable to cutaneous sensation may be grouped into—Hemianæsthesia ; Anæsthesia of the lower half of the body ; Localised anæsthesia ; Hyperæsthesia ; Paræsthesia and perverted or subjective sensation. Syringomyelia may be appropriately described in this place.

The CLINICAL INVESTIGATION is given in Part B. ; time and patience are required to elicit the precise nature of the defect and its boundaries. All three kinds of sensation may be lost ; tactile and painful generally go together, thermal may be lost alone. Muscle sense is not connected with the skin ; it is the sense of position or degree of contraction of a muscle.

I. **Hemianæsthesia**, or loss of tactile sensation in one half of the body, may be due to either functional or organic causes, the former being far the most frequent.

1. In **hysteria**, anæsthesia, and particularly hemianæsthesia, is such a frequent symptom as to form one of the hysterical stigmata. Hysterical hemianæsthesia has special characteristics of its own, which enable one to recognise the condition from these alone. (i.) In its typical form it is complete, more absolute than any other hemianæsthesia, and involves not only the skin and mucous membranes but also the joints and muscles. (ii.) It is sensorial, *i.e.*, involving the special senses, as well as common sensation. Vision alone may be partially retained on the paralysed side, but in its characteristic form this sense also is altered by a contracture of the field of vision on both sides, more marked on the anæsthetic side ; there is also an inversion of the colour fields.[1] (iii.) The hemianæsthesia varies from day to day, and perhaps from hour to hour, and may shift suddenly to the opposite side after some emotional shock. It may be accompanied by paresis of varying degree on the same, or the opposite side ; or the other side may be hyperæsthetic. (iv.) The condition may also be recognised by the presence of the other three hysterical stigmata, viz. globus ; fainting or nervous attacks ; and the ovarie. This latter consists of a hyperæsthetic patch in the inguinal region, which is known as the ovarian tenderness, though it is wholly independent of the ovary, for it is present in the male.

2. **Organic lesions of the brain or spinal cord** may also give rise to hemianæsthesia, though it is usually less profound than the preceding, and is accompanied by paralysis of the same side.

(a) Hemianæsthesia, sensory and sensorial, due to a very extensive organic lesion of the *cortex*, is recognised by convulsions and paralysis having a distribution corresponding to the position of the lesion.

(b) Hemianæsthesia, sensory and sensorial, of a more complete kind than the preceding, may be due to a lesion at the posterior end of the *internal capsule* (sensory crossway) and is known by (i.) a visual defect consisting of a hemianopsia, and (ii.) the leg is usually more involved than the arm because, it will be remembered, the leg fibres pass down the posterior part of the motor tract, adjacent to the sensory crossway.

(c) Hemianæsthesia without loss of sight or smell may arise from a focal lesion below the internal capsule but above the pons—a small hæmorrhage into the *tegmentum of the crus*, for instance.

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[1] *E.g.*, a case recorded in the "St. Thomas's Hospital Reports, 1888."

(d) Crossed hemianæsthesia (one side of body, other side of face) may arise from a lesion in the *fillet* or *pons*, in which case it is attended always by crossed hemiplegia. Crossed hemianæsthesia may also arise from damage to one of the *peduncles of the cerebellum*, especially the middle one, and is then accompanied by paresis from pressure on the motor strands, and is probably accompanied by staggering gait, vertigo, etc.

II. **Anæsthesia** of the lower half of the body or loss of tactile sensation of the legs and lower part of the trunk is very rarely functional. It indicates practically in all such cases an organic lesion of the cord, and is accompanied by paralysis. The segment of the cord involved may be localised by the upper limit of the anæsthesia. It will also be remembered that in myelitis a narrow band of hyperæsthesia is a good indication of the upper limit of the mischief, which may also be localised by means of Head's algesic areas (§ 551).

Brown-Séquard paralysis. An injury or lesion confined to *one half* of the cord produces paralysis in the leg of the same side, anæsthesia of that on the opposite side.

III. **Localised or limited anæsthesia.** (1) *Hysteria* is the commonest cause of complete SEGMENTAL anæsthesia of one limb, *i.e.*, one which is limited by a horizontal ring round the level of a joint (wrist, elbow, knee, or shoulder). Such an anæsthesia is always cerebral in origin.

(2) *Tabes dorsalis* is a disease which may be suspected when there are small PATCHES or streaks of lost or modified sensation. All manner of sensory variations are met with in the preataxic stage, such as anæsthesia of the soles of the feet or along the ulnar margin of the wrist, delayed sensation, a zone of an- or hyper-æsthesia round the body, or transferred sensation (allocheiria).

(3) *Syringomyelia* is a spinal lesion characterised by muscular atrophy and THERMAL ANÆSTHESIA of one or more extremities, not infrequently the arms, tactile and pain sense being retained (§ 601).

(4) Lesions affecting the *spinal nerve roots* (*e.g.*, spinal caries or tumours) produce PATCHES of anæsthesia along the limbs, and are accompanied by severe neuralgic pain. A transverse lesion of a *spinal segment* not involving the spinal roots is unaccompanied by pain. The distribution of the anæsthesia is given in the figures in § 551, and it differs from that due to peripheral nerve lesions.

(5) Peripheral neuritis and destructive lesions of the *peripheral nerves* also produce similar patches of anæsthesia in their areas of distribution (figs. § 564); but it should be remembered that loss of sensation is produced much less readily than loss of motion. In multiple peripheral neuritis there is very often analgesia with preservation of the tactile sense quite at the ends of the limbs. In lesions of the nerve trunks (neuritis, leprosy, etc.), the distribution of the anæsthesia corresponds only roughly with the distribution of the affected nerve, the adjacent areas overlapping considerably. But in lesions of a spinal root or of a spinal segment the area of anæsthesia does not correspond with the distribution of the nerves connected with that root; and the margins of the analgesic areas are abrupt and definite (see Head's areas, § 551).

Anæsthesia dolorosa is a condition of anæsthesia attended by neuralgic pains, present sometimes in peripheral neuritis, local or multiple.

IV. **Hyperæsthesia.** Much that has been said about anæsthesia applies also to hyperæsthesia, for irritative lesions will produce this latter condition where destructive lesions produce anæsthesia.

General hyperæsthesia occurs in hysteria, hydrophobia, and rickets. It is also found combined with the neuralgic pains in spinal meningitis (especially the acute form).

Localised hyperæsthesia. In alcoholic or other multiple neuritis the muscles, nerves and skin are often acutely tender. The band of hyperæsthesia in myelitis associated with girdle pain has already been referred to; it is a marked feature in cancer of the vertebræ, and in acute spinal caries.

In neuralgia *hyperæsthetic spots* on the skin are met with in the distribution of the affected nerve.

Tender areas may be associated with irritation of certain spinal root zones, or with visceral disease (compare § 551, Head's sensation areas). But perhaps the most common cause of hyperæsthetic spots is hysteria. Such spots are found very constantly in hysterical cases in the inguinal (so-called ovarian), inframammary and sometimes in other regions. If pressure over these spots gives rise to hysterical attacks of any kind, they are called hysterogenic zones.

V. **Paræsthesia** (perverted sensation) in its strictly etymological sense means the perverted perception of an external stimulus. Used in this sense it is met with either in the form of (i.) *transferred* or misplaced sensations (allocheiria), when the patient locates a tactile sensation to the opposite side or some other part of the body, or (ii.) *delayed* sensation. Both these alterations are met with in tabes dorsalis; and they may also be present in hysteria, and sometimes in syringomyelia. *Polyæsthesia* indicates an apparent multiplication of the parts touched. But the term paræsthesia is also frequently used for various subjective sensations independent of external stimuli, such for instance as the numbness, tingling, creeping cold sensations which are met with in the early stages of peripheral neuritis.

Neurasthenia is frequently accompanied by a number of perverted sensations, such as burning, crawling, throbbing, etc. In this disease also we meet with those indescribable *somatic sensations*, in which, for example, the patient feels as if he were "sinking through the earth," "treading on air," or a general feeling of bodily discomfort, such as words fail to describe.

Acroparæsthesia also comes under this head. It is a symptom but little referred to in books, but is common enough in out-patient practice. The patients complain of a feeling of coldness, pricking, smarting, pins and needles and often severe pains in the hands. It is a symptom of many nervous and gastric disorders and may go on to erythromelalgia (§ 430).

§ 601. Syringomyelia and intra-medullary tumours within the spinal cord. Tumours within the spinal cord consist mainly of syphilitic gummata, syringomyelia with or without gliomatosis, sarcoma, tubercle and myxoma. They are less frequent than the external and extra-medullary tumours (see compression paraplegia, § 550), and give rise to a very different set of symptoms. They involve the grey matter and the central parts of the cord primarily, consequently paraplegia and pain are usually absent until late in these diseases and only arise as a result of the secondary lateral sclerosis. *Syringomyelia* is an interesting condition which has received considerable attention of late years. It consists essentially of a patency or dilatation of the central canal of the cord, the epithelial lining of which is prone to take on a gliomatous (small-celled ? sarcomatous) growth having a malignant tendency to spread upwards and downwards. The chief seat, and therefore presumably the origin of this new growth is the cervical region, and consequently one or both arms exhibit the earliest symptoms.

The *symptoms* of these conditions vary somewhat with the position and the part of the cord involved (spinal localisation, § 551), but the commonest position is the anterior part of the posterior columns in the cervical enlargement, and we meet therefore with three principal symptoms:—(1) Loss of sensibility to thermic impressions and sometimes to painful impressions, tactile sensations being sometimes retained, chiefly but by no means always limited to the hands and arms, is the most characteristic symptom, and is not known to occur so typically in any other disease. Thermic, painful and tactile sensations are all translated simply as touch. (2) Muscular atrophy in one or both arms occurs as the tumour extends forwards, accompanied by R. D. (3) Later on there are paraplegic rigidity, increased reflexes and spasms due to descending sclerosis. In one case under my care there was atrophic flaccid paralysis of the legs with greatly increased knee-jerks and ankle-clonus. Diminished power of the limbs on one side with diminished sensation on the other (Brown-Séquard phenomenon) may be met with if the lesion extends laterally. Combined with the thermal anæsthesia and atrophy in the arms and the spastic paralysis in the legs are a number of other symptoms which may or may not be present, such as various trophic lesions of joints and bones. At other times there are vesicles, bullæ, extensive desquamation of the cuticle (in one case under my care the epidermis of the fingers was shed like a glove), ulcerations or whitlows (which coming on without pain resemble the painless whitlows of Morvan's disease), all of which chiefly affect the upper extremities. When the lesion spreads to the medulla and pons the cranial nuclei are apt to become involved, causing nystagmus, deranged respiratory and cardiac movements, paralysis of the vocal cords, tongue, palate or pharynx. Sudden death may ensue as in the case just mentioned from extension to the "nœud vital." These are the symptoms of tumours in the cervical region and of syringomyelia: in the other positions intra-medullary tumours give rise to symptoms which depend on the column mainly involved.

The *diagnosis* is difficult in the earlier stages. The pathognomonic symptoms of syringomyelia are thermal anæsthesia combined with atrophic paralysis. Progressive muscular atrophy has no such impairment of sensation. In multiple peripheral neuritis the symptoms are more scattered. Hypertrophic cervical meningitis is characterised by severe pain, and the anæsthesia if present involves all forms of sensation.

The *prognosis* depends upon the position and rate of advance of the disease. Syringomyelia usually runs a very slow but progressive course of many years unless the neoplasm extends rapidly upwards and causes death. Other cases in which apparently no gliomatosis occurs, and in which some joint[1] or other trophic manifestations are the only symptoms, may live to old age.

Causes. Syringomyelia is probably due to a congenital condition of the cord, but the age at which the symptoms are first noticed is not often under 15 and very rarely over 30. It is more frequent in males (two-thirds). The symptoms have often been noticed to follow an accident of some kind. The central canal (so-called) of the cord consists normally of a solid rod of epiblastic cells remaining from the grooved infolding of that structure which develops into the spinal cord. Everything tends to prove that this remains patent in syringomyelia and that the cavity enlarges and involves the adjacent trophic and sensory tracts, especially that of the temperature sense. In some cases the condition remains stationary and the patient lives indefinitely; in others gliomatosis occurs in the epiblastic cells (determined perhaps by a slight injury to the back) and the patient dies in a year or two. Or, syringomyelia may consist of multiple cavities in the cord with or without gliomatosis, these affording a predisposition (Gowers says a necessary predisposing factor) to hæmorrhage into the cord.

The *treatment* is purely symptomatic, though iodide may be tried, and Coley's fluid, if we have reason to believe that the central growth is of a malignant character.

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[1] As in a case exhibited by Mr. J. R. Lunn at the Clinical Society of London in 1899 or 1900.

GROUP VI. CRANIAL NERVES AND SPECIAL SENSES.

The investigation of the cranial nerves and special senses is of great importance from the standpoint both of general medicine and neurology. Here is a tabular statement of their function.

TABLE OF CRANIAL NERVES AND THEIR FUNCTIONS.

Cranial Nerves.	*Functions.*
I. Olfactory nerve.	Smell.
II. Optic nerve.	Sight.
III. Motor oculi	Supplies all the *muscles* of the eyeball (except the superior oblique and external rectus) and the levator palpebræ superioris; also the sphincter pupillæ and ciliary muscle.
IV. Motor oculi.	Supplies the superior oblique; turns the eye down and outwards.
VI. Motor oculi.	Supplies the external rectus; turns the eye outwards.
V. Trigeminal nerve. 1st division, Ophthalmic.	*Sensory* to forehead and part of vertex, anterior part of nose to tip, upper eyelid and temple, eyeball and lachrymal gland. Contains dilator pupillæ fibres from sympathetic.
2nd division, Superior maxillary.	*Sensory* to cheek, lower eyelid, side of nose and upper lip; the upper teeth and gum; lining membrane of nose, roof of mouth, soft palate, tonsils and roof of pharynx. *Taste* of anterior $\frac{2}{3}$rds of tongue (through Maeckel's ganglion by chorda tympani nerve). *Trophic* and vaso-motor fibres.
3rd division, Inferior maxillary.	*Sensory* to lower part of face, lower lip, side of head, ear, tongue, lower teeth, gum and inner side of cheek. *Motor* to masticatory muscles, temporal, masseter, pterygoids, anterior belly of digastric and mylo-hyoid, tensor tympani and ? tensor palati. *Taste* of anterior $\frac{2}{3}$rds of tongue (by chorda tympani from lingual nerve); of posterior $\frac{1}{3}$rd of tongue (through glosso-pharyngeal nerve, Jacobson's nerve and otic ganglion).
VII. Facial nerve.	*Motor* to all muscles of face and scalp (excepting levator palpebræ superioris), platysma, posterior belly of digastric, and stapedius muscle. It is joined by the chorda tympani (conveying taste fibres of anterior $\frac{2}{3}$rds of tongue from lingual branch of V. to Maeckel's ganglion).
VIII. Auditory nerve.	Hearing.
IX. Glossopharyngeal nerve.	*Sensory* from pharynx. Collects taste fibres from posterior $\frac{1}{3}$rd of tongue, which ultimately join V. *Motor* to middle constrictor of pharynx and stylo-pharyngeus.
X. Vagus nerve.	*Motor* for soft palate (except tensor palati), pharynx and larynx (through accessory portion of XI.). *Motor* (involuntary) and *sensory* for heart, respiratory passages and abdominal viscera (through sympathetic ganglia).
XI. Spinal accessory nerve.	*Motor* to sterno-mastoid and trapezius. (Supplies vagus with motor fibres for larynx, pharynx and palate.)
XII. Hypoglossal nerve.	*Motor* to tongue and depressors of hyoid bone.

§ **602.** The **first nerve** is the olfactory bulb. To test the power of smell on one side close the other nostril with your finger and tell the patient to sniff some odour such as peppermint, clove oil, or onions (not ammonia, which is a stimulant to the second division of the fifth nerve in the nasal mucous membrane), and see if he can name the odour. It is convenient to have some little bottles containing these substances.

Anosmia is loss of smell; *parosmia* is a perversion of smell. Loss of smell often depends on atrophic rhinitis or some other nasal disorder (see Chapter VII.). The first nerve alone may be damaged by concussion, the fine branches becoming torn against the cribriform plate of the ethmoid. This is extremely difficult to restore; in one case I was partially successful by localised cataphoresis with cocaine and strychnine. Smell may be lost after influenza, diphtheria and other specific fevers, after thrombosis or embolism of the anterior cerebral artery, and with tabes dorsalis. Paralysis of the fifth may produce it by dryness of the nostrils. Hallucinations of smell, *i.e.*, odours recognised when no odorous substance is present, often form the aura of an epileptic fit.

§ **603.** The **organ of vision** is innervated mainly by four cranial nerves—the *second*, *third*, *fourth* and *sixth*. The *fifth* and the cervical *sympathetic* are also concerned in its innervation. Careful examination of the eye is of the greatest importance in many diseases.

The symptoms which reveal disease of the eye may be arranged under 6 headings—Pain, Superficial Alterations, Acuteness of Vision (§ 604), Pupils (§ 605), Ocular Movements (§ 606), Changes in the Fundi (§ 607). The reader should turn to the section dealing with the defect to which the patient's symptoms appear to belong.

The systematic examination of the eye consists of (1) noting any superficial alterations; (2) testing the acuteness of vision; (3) examining the pupils; (4) the ocular movements; and (5) the fundi. But in routine examination for medical purposes the acuteness of vision is left until the end.

I. **Pain in the eyes** is not infrequently absent in ocular affections. Its commonest cause is some error of refraction (asthenopia, *i.e.*, eye-strain). Eye-strain may cause a dragging pain round the eyes, or blepharitis and blinking in children, or severe neuralgia of the fifth nerve. Glaucoma or any other cause of increased ocular tension may cause pain. And as Tomes[1] has pointed out, dental disorders may not only give rise to reflex pain but also to both functional and organic affections of the eye. Among subjective sensations other than pain may be noted muscæ volitantes (black dots) and scintillating scotoma (zigzag lines); the former occur in anæmia, debility, or functional disorders of the liver, the latter in association with migraine.

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[1] "A System of Dental Surgery," Charles S. Tomes, F.R.C.S. London, 1897, p. 572 *et seq.*

II. **Superficial alterations** mainly come within the province of the surgeon; they are 6 in number. A good light and a lens are all that is necessary.

1. Proptosis is an undue prominence of the eyeballs, and is met with in exophthalmic goître, ophthalmoplegia externa, and in marked myopia. In one eye only it suggests orbital tumour or some intracranial lesion. To detect small degrees look down over the top of the patient's head from behind. Recession of the balls occurs in paralysis of the cervical sympathetic, the other symptoms of which are contraction of the pupil on that side and loss of the cilio-spinal reflex (reflex dilatation of the pupil when the skin of the neck is pinched).

2. The eyelids. The eyelids are puffy in renal disease, cardiac dropsy, and the arsenical poisoning. Ptosis, or drooping of the upper eyelid is (i.) one of the symptoms of paralysis of the third nerve (*infra*). It may also be due to (ii.) a cortical lesion, without involvement of any other part of the 3rd nerve; (iii.) idiopathic muscular atrophy when the face is affected; (iv.) myasthenia gravis; (v.) hysteria; (vi.) a congenital condition; (vii.) Nothnagel has described a pseudo-ptosis, in which the eyelid gives the appearance of drooping owing to recession of the eyeball in paralysis of the cervical sympathetic (§ 605). A diminution of the palpebral fissure may be due to recession of the eyeball (*ante*), or weakness of the levator palpebræ superioris. Blepharospasm is an involuntary clonic twitching of the eyelid (see habit spasm, etc.). Inability to close the eye is a notable feature in Bell's (facial) paralysis. It is also one of the earlier symptoms, in infancy, of the hereditary infantile amyotrophy described by Duchenne.

Deficient falling of the upper eyelid when the patient looks rapidly down constitutes the so-called Von Graefe's sign in exophthalmic goître, but this symptom is only present in an advanced stage of the disease when other means of identification are not wanting. The same remark applies also to Stellwag's sign (the almost total absence of involuntary blinking), and to Mœbius's sign (a deficient power of convergence), which are occasional symptoms in Graves' disease.

The diseases of (3) the conjunctiva, (4) the cornea, (5) the iris and (6) the alteration of tension are dealt with in surgical works.

§ **604.** III. **Defects of vision** may consist of (1) defective sense of form or acuteness of vision, (2) alteration in the field of vision, (3) defective sense of colour.

(1) Acuteness of vision implies the estimation of forms of objects. It may be roughly tested by asking the patient to count the number of fingers held up before him. The defect may be so great that the patient cannot perceive light from darkness. The eyes must be examined separately, as it is often found that defect of one eye has existed a long time without the patient being aware of it. If the external parts of the eye are normal, the media transparent, the ophthalmoscope reveals no disease and there is defective acuity of vision, it is probable that the patient suffers from an error of refraction. Asthenopia (eye-strain) is due to continuous overaction of the internal muscles of the eye, and is manifested by ocular pain, ciliary congestion and headache. Excessive blinking and

inability to use the eyes for long at a time are often the only symptoms in children. It may be due to overuse of the eyes in a bad light, but its commonest cause is some error of refraction.

ERRORS OF REFRACTION. For accurately testing the visual sense of form the power of reading Snellen's types at a given distance (usually 6 metres or 20 feet) is employed. The error of refraction is ascertained (after paralysing the iris by homatropin) by placing various lenses in the frame-spectacles before the eyes until it is found which of them completely corrects his error. Convex lenses are indicated by the sign +, concave by the sign −. The defect is measured by the focal length of the lens required to correct his error, and is now generally expressed in diopters, indicated by the sign D. A lens of one diopter has a focal length of one metre. Thus a + 3 D lens indicates a convex lens with a focal length of $\frac{1}{3}$ metre, being three times as strong as a lens of + 1 D. Retinoscopy is a more accurate method of testing refractive errors (below). In *myopia* (or near sight) the image is formed in front of the retina, and the patient cannot see distant objects clearly: in *hypermetropia* (or far sight) the image is formed on a plane behind the retina, and the patient cannot look at near objects for any length of time. Both may be due to either defective shape of the globe or to defective accommodation. Concave lenses are used to correct myopia, and convex to correct hypermetropia. In *presbyopia* the rigidity of the lens renders it either difficult or impossible to accommodate for near objects; it occurs in old people whose far vision may be remarkably good, though it is impossible for them to read or to see near objects distinctly without convex glasses. *Astigmatism* is a non-correspondence of the curve in the different meridians of the cornea. In *simple* astigmatism one meridian is normal, the ether myopic or hypermetropic; in *compound* astigmatism the error of the two meridians, though of the same kind, differs in degree; in *mixed* astigmatism there is a myopic error in one meridian, and a hypermetropic error in the other meridian; in *irregular astigmatism* the curves of the cornea vary even in the same meridian. Astigmatism is detected accurately by a skilled examination by retinoscopy, or with the astigmometer.

In **retinoscopy** the procedure is as for indirect ophthalmoscopy (§ 607), only without the large lens. No details of the fundus are visible in this way, but normally a perfectly red field is seen. If the blood vessels are seen, then the patient's refraction is abnormal. *Errors of refraction* are revealed by tilting the mirror upwards and downwards and from side to side. A black shadow passes across the field in this procedure. In a normal eye the shadow moves very rapidly, and has a straight, defined edge; the greater the error of refraction the more slowly does the shadow move, and the more curved and hazy is its margin. If the edge of this shadow moves in the opposite direction to the mirror, the refraction is either normal, hypermetropic, or consists of less than one D. of myopia. If there be myopia of more than one D., the shadow moves in the same direction. Astigmatism is discovered by the shadows moving differently in opposite meridians. *Opacities* in the media may be thus detected as dark shadows upon the red field. The radiating streaks of commencing cataract, or moving opacities in the vitreous may also be thus detected.

(2) The FIELD OF VISION is the extent of the picture presented to the eye at any given moment. It may be roughly tested by instructing the patient to cover one eye and look fixedly at the tip of your nose at a distance of about 2 feet. Then hold up one hand on each side of you in the same vertical plane as your face, and bring it gradually towards your nose, asking the patient to cry "stop" the moment it comes into his view. Repeat the same procedure above, below, and at the two sides, and in this way you will roughly ascertain in what part of his field the vision is defective. The dimensions of the visual field can be tested accurately only by the perimeter (below). *Scotoma* is a word used to indicate a spot

of blindness or imperfect vision within an otherwise healthy field, *e.g.*, a central scotoma is a blind spot in the middle of the visual field.

The perimeter. The patient covers one eye and places the other cheek bone against the round knob of the vertical pillar half an inch below the palpebral fissure. The patient must be educated to keep his eye steadily fixed on the spot opposite, while the operator moves a small piece of white paper (or coloured for testing colour vision) mounted on the stick provided along the movable semi-circular metal band from periphery to centre. The position in which the patient can first see the paper (while looking fixedly all the time at the central spot) is then marked on the chart provided. With stupid patients this is a tedious operation, and without due care erroneous results may easily be obtained. The perception of colours in the peripheral field varies normally in extent with the different colours. Thus, from without inwards they are white, blue, yellow, red, green.

(3) COLOUR VISION may be tested by means of a collection of different coloured wools. The patient should be instructed either to (i.) identify and name different colours, or (ii.) to pick out all the pieces of wool which match, 3 or 4 pattern pieces being given to him at the outset.

Colour blindness (achromatopsia) is a symptom in some diseases of the retina and optic atrophy, both primary and secondary. It is also present in tobacco and some other forms of amblyopia in their early stages (*infra*). Partial achromatopsia may be congenital. In some hysterical cases the colour defect takes the form of dyschromatopsia, where the fields for different colours are inverted, the field for red being larger than that for blue, the reverse of normal.[1] Charcot,[2] who was the first to describe this condition, regarded this as a special and quite distinctive feature of hysterical achromatopsia. *Coloured vision* (chromatopsia) is an occasional symptom after the extraction of cataract in aged persons, and in exhausted states of the nervous system. I have met with it in some cases of tabes dorsalis. It is also occasionally a symptom of glaucoma; red vision (erythropsia) is the most common. Yellow vision is a symptom of santonin poisoning.

The **causes of defective vision** without any very obvious ocular changes may be considered under amblyopia (bilateral and unilateral), hemianopsia, and night-blindness. The defective vision due to errors of refraction has already been dealt with.

AMBLYOPIA is diminished vision, AMAUROSIS loss of vision, without discoverable changes in the fundi oculorum or error of refraction. It is obvious that amblyopia may be due either to some functional disturbance of the visual apparatus, or to some gross lesion of the brain or paths of vision behind the retina or optic discs.

BILATERAL AMBLYOPIA may arise under various toxic conditions and functional states, as follows :—

1. *Tobacco amblyopia* arises sometimes in hard smokers of over 3 or 4 ozs. per week, or in debilitated persons from a much less quantity. The patient first complains of defective vision in bright light ; he sees better at dusk than at noon. The defect is slowly progressive, becoming most marked in the *central* part of the field, and there is central colour scotoma. At first there may be no changes in the fundi ; then the discs become slightly congested in the earlier stages, and pale and atrophied, especially on the temporal side, in the later. In several cases which I have seen, a defective colour vision was the earliest symptom to attract the patient's notice. Tobacco amblyopia, it should be borne in mind, is very occasionally met with in females.

2. *Toxic* amblyopia is caused also by other poisons such as uræmia, diabetes, large doses of quinine, bisulphide of carbon in indiarubber manufacture, iodoform and dinitro-benzol. Little in the way of treatment can be done for such cases unless they are seen early, when absolute cessation of the cause, and functional rest to the structures involved may lead to recovery.

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[1] A case is recorded in "St. Thomas's Hospital Reports, 1888."

[2] "Clinical Lectures on Diseases of the Nervous System," vol. iii. ; New Syd. Soc. Trans.

3. Various *structural diseases of the retina and optic nerve* in their incipient stages, when attended by such slight alterations that none but a very skilled observer can detect, may nevertheless be attended by an alteration of the visual fields and of the acuteness of vision. This is sometimes the case with insidious papillitis and generally with primary optic atrophy. The double primary atrophy which so often accompanies tabes dorsalis is a marked instance of this; it may precede the ataxy and other symptoms of tabes by many years. I have seen several such cases, where the amblyopia was regarded as functional, and its spinal origin overlooked for a long time. Primary atrophy more rarely accompanies disseminated sclerosis and G. P. I. In all these cases the field of vision is diminished, either eccentrically or concentrically, and colour vision also is reduced.

4. *Hysterical amblyopia* is a very characteristic affection, and consists of a concentric retraction of both fields, though most extreme on the same side as the hemianæsthesia by which it is almost invariably accompanied. The colour vision is also affected in a characteristic manner (see *ante*).

5. *Neurasthenic* or *retinal asthenopia*, a condition in which the patient ceases to be able to see after exercising the visual function for a time, is also unattended by changes in the fundi. The field of vision is also contracted, and temporary scotomata appear on looking at objects.

6. *Sudden and copious hæmorrhage* (*e.g.*, from the stomach, bowels, uterus, etc.) may produce amblyopia from deficient blood supply to the eyes, and possibly to the visual cortical centres. If it does not shortly clear up, optic neuritis is to be apprehended.

7. Exposure to *blinding sunlight* or electric light may cause acute amblyopia.

MONOCULAR AMBLYOPIA. It must be borne in mind that defective vision in one eye may be overlooked for many years, the patient thinking that he suddenly becomes blind when he has by chance shut the good eye on one occasion.

1. The remarks made above relative to *optic neuritis* and *atrophy* apply here. Local diseases of the choroid or retina in the macular region produces central amblyopia, or central scotoma, often of one eye only.

2. *Squint*, in some cases, produces a form of congenital amblyopia in childhood, which is only very slowly, if ever, remedied. It happens thus: squint results in double vision, and in order to see clearly, the child unconsciously gets into the habit of neglecting the image produced by one of the two eyes, usually the squinting one. After long habit this "suppression of image" becomes permanent. It would perhaps be more correct to say that the mind has lost the power of perceiving the image formed in that eye, and can only be slowly educated up to it.

3. *Astigmatism, or my- or hypermetropia* in a very high degree (where a clear image may never have been formed on the retina) leads to defective vision, so that when a full correction of error of refraction takes place, it is generally found that the patient cannot see. This probably arises from the fact that the retina has never received the necessary practice or education for the appreciation of true images of objects.

4. *Chill.* A few cases appear to be produced by exposure to cold. Occurring for the most part in young adults, the onset is fairly rapid, and accompanied by neuralgic pains in the same side of the head. The

affection is not usually serious, and recovers under the same means as those employed for paralysis of the facial nerve under like circumstances.

5. *Reflex amblyopia* is certainly not common. It generally occurs only in one eye, and is associated with a carious tooth. Sir Thomas Watson[1] mentioned cases, and quite a number of cases have been collected by Tomes,[2] in which the blindness was cured by the adequate treatment of a decayed tooth, and returned when this again gave trouble.

HEMIANOPSIA (hemianopia, hemiopia) means loss of sight in one half of the visual field in both eyes, unaccompanied by changes in retina or disc. Four kinds are described, but practically the first is the only one met with, and that is sufficiently rare.

Homonymous or lateral *hemianopsia* means loss of vision of the two left or right halves (the corresponding halves) of the visual fields of each eye. Right lateral hemianopsia means abolition of the right halves of the patient's visual fields. This corresponds to the temporal half of the left and the nasal half of the right retina (see Fig. 167). Apart from its occasional occurrence in migraine, it is due to a gross central lesion situated in some part of the visual path behind the chiasma—(i.) in the optic tract, or (ii.) behind the corpora quadrigemina, that is to say, the hinder end of the internal capsule or the white fibres of the occipital lobe, or (iii.) in the visual centres of the cortex, situated in the occipital lobe. By employing Wernicke's test (§ 605) the first may be excluded. For the rest, the precise position and character of the lesion can only be diagnosed by the accompanying symptoms. Cortical lesions are usually associated with some form of aphasia, or with paralysis due to some cerebral lesion. Lesions affecting the optic tract are syphilitic gummata or meningitis, tubercle and neoplasmata, softening and hæmorrhage (rare). Tumours in the optic thalamus, lenticular nucleus and temporo-sphenoidal lobe may extend to or press upon the optic tract. Other associated basal paralyses may aid the diagnosis.

The remaining variety (*heteronymous* hemianopsia) is vary rare; it may occur on one side only. *Temporal hemianopsia* is the loss of the outer half of each field, due to a blindness of the inner half of each retina. In the only cases on record it has arisen from damage to the middle of the optic chiasma, or rather, just behind or just in front of the middle part. Pressure of a tumour, distended third ventricle, or localised basal meningitis have produced it. It has been met with in some cases of acromegaly. Recurrent attacks have been produced by gummata. *Nasal hemianopsia* is still rarer. It is usually unilateral, due to a lesion situated in one optic tract just as it leaves the chiasma. *Altitudinal hemianopsia* means loss of the upper or lower part of the fields, and if bilateral is due to a lesion involving the upper or lower part of the chiasma, optic neuritis, or symmetrical cortical lesions.

NIGHT-BLINDNESS (nyctalopia) is defective vision in dim lights. It is a feature of retinitis pigmentosa, syphilitic retinitis, and it may be congenital (without fundal changes). Eccentric contraction of the visual field supervenes, and finally complete blindness. Acute night-blindness may attack those with defective general health who have been exposed to very strong sun or artificial light, and in these cases the prognosis is good.

§ 605. IV. **Defects in the pupils.** The iris comprises dilator fibres (supplied by the cervical sympathetic), and constrictor fibres (supplied by the 3rd nerve); associated with these latter is the ciliary muscle, also supplied by the 3rd nerve. Each of these may be paralysed separately, and paralysis of all three is known as *ophthalmoplegia interna.* The pupils must be tested in regard to their shape, size, equality, and mobility to light and accommodation. The patient should be placed opposite a good light.

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[1] "Lectures on the Principles and Practice of Physic," 4th edition.

[2] "A System of Dental Surgery," 4th edition, p. 574 *et seq.*

The *shape* of one or other pupil may be irregular, and this may be due to old iritic adhesions or the result of previous iridectomy. Coloboma iridis is a congenital abnormality which sometimes exists without error in vision, consisting of a deficiency of the iris, usually in the lower part, generally on both sides.

The *mobility* of the pupils must be tested both by the *light* and *accommodation* reflex (see below).

The average *size* of the pupils varies in healthy persons with the light and irritability of the retina and optic nerve. When two pupils are *unequal* in size and it is desired to ascertain which is the normal one of the two, it may generally be inferred that the immobile pupil is the abnormal one. Slight inequalities between the pupils may also be observed in health. Inequality of the pupils of the two eyes is a frequent symptom in G. P. I., and an occasional one in tabes and migraine. It may exist obviously in any condition causing contraction or dilatation of the pupil on one side only (see below), as for instance in paralysis of one 3rd nerve causing dilatation, or paralysis of the cervical sympathetic causing contraction.

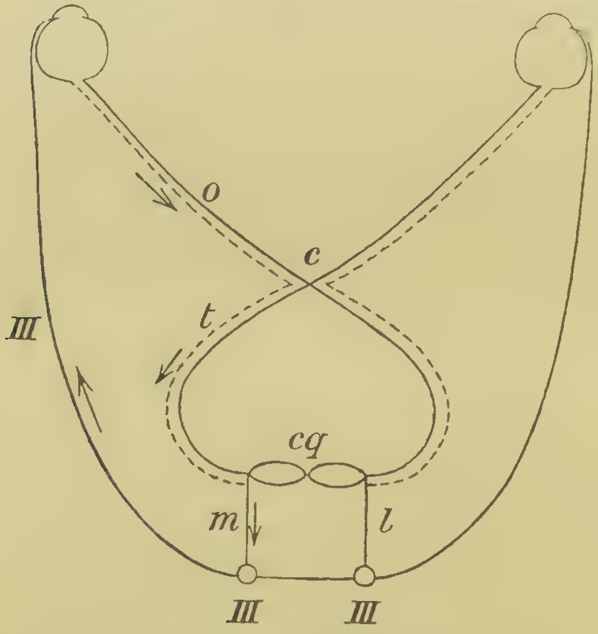

Fig. 167.—Diagram showing REFLEX ARCS concerned in the MOVEMENTS OF THE PUPIL (Horizontal Plane). *o*, optic nerves; *c*, optic chiasma; *t*, optic tract; *cq*, corpora quadrigemina; *III*, third nerves and nuclei; *m* and *l*, Maynert's fibres communicating between the third nuclei and the corpora quadrigemina. (Vertical plane fig. 168.)

The MOBILITY OF THE PUPILS to LIGHT. In testing the light reflex, *both* eyes should be covered for half a minute, and each uncovered in turn opposite to a bright light, which makes the pupils contract. In a good light the iris can sometimes be observed to contract and dilate rhythmically, a phenomenon which is called hippus. Its clinical significance is unknown. *The pupil light reflex* depends on the integrity of the retina and the following tract (Fig. 167), the optic nerve (*o*), the chiasma (*c*), optic tract (*t*), to the corpora quadrigemina (*cq*). These last named nuclei (*cq*) are connected, by means of Maynert's fibres (M), with the nuclei of the third nerves (III.) situated in the floor of the aqueduct of Sylvius. The fibres of the third nerve, through the long or short ciliary branches, conduct contracting impulses to the sphincter iridis.

Loss of light reflex (light iridoplegia) may be produced by a lesion situated anywhere in these afferent or efferent tracts. (i.) In tabes the pupils may be unequal, unduly contracted, or fail to react to light or accommodation, but the change most commonly met with is the Argyll-Robertson pupil, which is Loss of reflex to Light, though Acting to Accommodation. This phenomenon met with occasionally in other conditions indicates a partial damage involving either of the optic tracts or Maynert's fibres. (ii.) Atrophy of both optic nerves. (iii.) Destructive lesions of the third nerve or its nucleus. (iv.) General paralysis of the insane. The light reflex is preserved in blindness of central origin. In unilateral optic atrophy, when the sound side is uncovered both pupils contract (consensual contraction), but the pupil on the affected side does not contract when the affected side alone is uncovered.

Wernicke's pupil reflex assists one to determine the seat of a lesion in a case of hemiopia. Hemiopia may be due to a lesion (i.) of the optic tract between the chiasma and corpora quadrigemina (see Fig. 167); (ii.) of the optic fibres between the *cq* and the occipital cortex; or (iii.) of the occipital cortex. If the lesion be in the optic tract, the light reflex is lost, but if behind the *cq*, a beam of light thrown directly on the blind half of the retina by the concave mirror of the ophthalmoscope produces contraction of the pupils. A little study of Fig. 167 will make this apparent.

MOBILITY OF THE PUPILS TO ACCOMMODATION. Whenever the eyes converge and the patient looks at a near object, the pupils contract. Ask the patient first to look at a distant object, and after observing the pupils tell him suddenly to look at the tip of your finger held 6 inches in front of his face. This is really a reaction to convergence of the eyes. Loss of power of accommodation depends on paralysis or weakness of the ciliary muscle (cycloplegia) usually associated with paralysis of the sphincter of the iris. Loss of mobility of the pupil to accommodation is one of the earliest and most common symptoms in (i.) diphtheritic paralysis, and occurs on both sides. It is also seen in (ii.) belladonna poisoning, and (iii.) occasionally tabes and G. P. I. It is preserved in the Argyll-Robertson pupil (*vide supra*).

OPHTHALMOPLEGIA INTERNA is paralysis of the 3 internal muscles of the eye (the sphincter and dilator pupillæ and the ciliary muscle). The pupils are of medium size and immobile to light and accommodation. It constitutes an early symptom of diphtheritic and sometimes syphilitic paralysis, in either of which it is due to toxic affection of the oculo-motor nuclei. It is often associated with ophthalmoplegia externa (§ 606).

CILIO-SPINAL OR SKIN REFLEX is the dilatation of the pupil when the skin of the neck on one side of a healthy person is stimulated by stroking, pinching, or faradisation, and is due to the stimulation of the dilator fibres contained in the cervical sympathetic (see Fig. 168). The loss of this skin reflex may be observed occasionally in tabes and G. P. I., but is chiefly met with as one of the symptoms of paralysis of the cervical sympathetic.

CONTRACTION OF BOTH PUPILS (myosis) may be caused by (i.) undue retinal irritability, which may ensue from overuse of the eyes, and from hypermetropia. (ii.) It is also present in congestion of the iris from any cause, which possibly explains their contraction in mitral regurgitation. (iii.) Various drugs, such as opium, tobacco, eserine, or pilocarpin. (iv.) Tabes dorsalis, which is often attended also by loss of the light reflex (*supra*). (v.) General paralysis of the insane is sometimes attended by the same condition of pupils, the "pin-point" pupils, *i.e.*, strong contraction with sluggishness of movement. (vi.) Irritation of the third nerve as by pressure of tumour or the early stage of all inflammatory or meningeal affections; or irritation of the pupil contracting centre, *e.g.*, hæmorrhage into the pons causes a state of coma with contracted pupils which may distinguish it from hæmorrhage into other situations, opium poisoning being excluded. (vii.) Paralysis of the cervical sympathetic.

Paralysis of the cervical sympathetic causes contraction of the pupil [illegible] on one side only. The cervical sympathetic conveys *dilator fibres* to the iris, which, arising probably in the floor of the aqueduct, pass down the spinal cord to the cilio-spinal centre (Fig. 168) which is situated at the junction of the cervical and dorsal regions, thence outwards by the rami communicantes to join the sympathetic ganglia in the neck, and upwards along the carotid and cavernous plexuses to be finally distributed to the eyeball along with the ophthalmic division of the fifth. The symptoms of paralysis of the cervical sympathetic are (i.) contraction of the pupil of that side with absence of dilatation of the pupil on shading the eye or the instillation of cocaine; (ii.) abolition of the cilio-spinal reflex (*vide supra*); (iii.) some recession of the eyeball so that the eye looks smaller than its fellow; (iv.) slight drooping of the upper lid on that side due to paralysis of its unstriped muscular fibres; and (v.) absence of sweating on that half of the head and neck, and perhaps the upper extremity on that side, even after the administration of pilocarpin or other sudorific

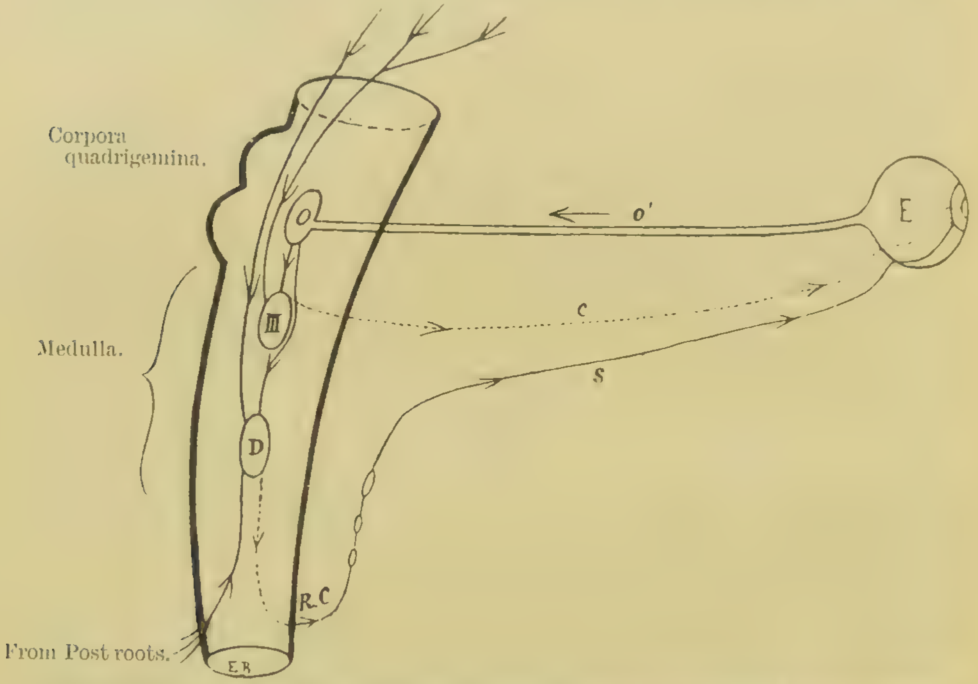

Fig. 168.—Diagram of the REFLEX ARCS concerned in MOVEMENTS OF THE PUPIL (vertical plane): *O*, nucleus of optic nerve in corpora quadrigemina; *O′*, optic tract, see fig. 167; *III*, nucleus of 3rd nerve (the upper part only being concerned in the pupillary movements); *C*, constrictor branches accompanying third nerve; *D*, dilator cilio-spinal centre; *S*, dilator fibrils from cervical sympathetic accompanying the ophthalmic division of the 5th; *R.C*, Rami communicantes of sympathetic through anterior spinal roots of 8th cervical nerve.

Paralysis of the cervical sympathetic may arise from the pressure of an aneurysm of the aorta or other intrathoracic tumour, from exostoses or other tumours in the neck, or from injury or disease of the spinal cord below the medulla but above the second dorsal segment.

DILATATION OF BOTH PUPILS (mydriasis) may be produced by—(i.) A condition of myopia. (ii.) The pupils are larger in childhood. (iii.) Certain drugs, belladonna, duboisin, cocaine. (iv.) Neurasthenia, and other exhausted states of the nervous system, *e.g.*, anæmia and typhoid. (v.) During an apoplectic or epileptic attack the pupils are dilated, and in such states it forms a valuable means of diagnosis, for the symptom cannot be feigned, and is present in all the genuine cases of coma, excepting from hæmorrhage into the pons. (vi.) In diphtheritic paralysis, bilateral dilatation of the pupil and loss of power of accommodation (cycloplegia) is an early and constant symptom, and may be accompanied also by complete ophthalmoplegia interna (dilator and sphincter iridis and ciliary muscle). (vii.) Paralysis of the third nerve (destructive lesions of the trunk or its nucleus) a condition which gives rise to 4 symptoms, dilatation of the pupil, loss of accommodation, external strabismus (producing diplopia), and ptosis. (viii.) Irritation of the cervical sympathetic as by spinal growths or meningitis. (ix.) Certain psychic emotions such as fear, and deep inspiration, dilate the pupil. Unilateral recurrent mydriasis often precedes mental derangement.

§ 606. V. Oculo-motor defects. The external muscles of the eyeball (as distinct from the internal or involuntary muscles of the iris) are 6 in number, and they are supplied by 3 cranial nerves—external rectus (VI. nerve), superior oblique (IV. nerve), internal, superior, and inferior recti and inferior oblique are supplied by the III. nerve (which also, it will be remembered, supplies the levator palpebræ, the contractor fibres to the iris, and the ciliary muscle of accommodation). It follows therefore that—

Complete paralysis of the 3rd nerve is attended by	Ptosis ; External strabismus ; Pupil dilatation and immobility ; Loss of accommodation ; Inability to move eyeball inwards or upwards, and only imperfectly downwards ; Eyeball slightly protruded.
Paralysis of the 6th nerve is attended by	Internal strabismus ; Inability to move eye outwards ; Homonymous diplopia.
Paralysis of the 4th nerve is attended by	Slight deviation of cornea upwards ; Homonymous diplopia on looking downwards.

Defects in the ocular muscles are revealed—(1) by defective movements of the eyeball ; (2) by squint ; (3) when the defect is only slight, by double vision (diplopia) ; (4) by ophthalmoplegia externa ; or (5) by nystagmus. The *first* question to answer is which of the muscles is affected ? The *second* question relates to the position and nature of the lesion.

(1) The **movements of the eyeballs** may be tested in simple cases by fixing the patient's chin with one hand and asking him to follow with his eyes your other hand moved slowly inwards, outwards, upwards, and downwards. Notice any deficiency of movements, jerkiness, or nystagmus. One eye may be tested at a time. The power of convergence should also be tested by asking him to look steadily at the tip of your finger while it is moved from a distance of 18 inches up to the tip of his nose.

Deficient movement of the front of the eyeball—	*indicates paralysis of*
outwards	external rectus—6th nerve.
inwards	internal rectus—3rd nerve.
upwards	*superior* rectus *inferior* oblique } 3rd nerve.
downwards	*inferior* rectus—3rd nerve. *superior* oblique—4th nerve.
downwards and outwards	superior oblique—4th nerve.

(2) **Squint** or strabismus is a want of parallelism between the two visual axes due to defect in one of the muscles. It is called *convergent* when the eyeballs both look inwards, and *divergent* when they look away from each other. *In children* it is mostly caused by the weakness of one muscle or overaction of the other, or both combined, and is due to some error of refraction—hypermytropia, with concomitant internal strabismus (the commonest in children), or myopia, with external strabismus. In adults squint is more often due to definite paralysis of an ocular nerve and the matter is of much greater significance (see below). In CONCOMITANT (or spasmodic) squint, the affected eye follows the sound eye with equal defect in all directions, that being the reason why it is called "concomitant" squint. Each eye, when the other is covered, moves perfectly in all directions, but when examined together, the squint is present in all positions of the eyeball, especially when the eye is looking straight forward, which is the position of rest for all the healthy muscles. The defect of parallelism remains the same in all positions. PARALYTIC squint disappears when the eye is directed straight forward, which is the position of rest, and only appears when the eye is moved into that position which necessitates the use of the paralysed muscle. Diplopia is present in paralytic, but not in concomitant squint.

To examine for squint and *to detect whether it is due to paralysis or spasm* and which is the affected muscle, the patient is told to look at an object straight in front of him, that being the normal position of the eyes at rest, and to fix some object. The eye with which he fixes is the normal eye. The deviation of the affected eye from the middle line is known as the "primary deviation." Now partially cover the sound eye and let him fix with the affected eye; the sound eye will be found to deviate ("secondary deviation"). In concomitant squint the primary and secondary deviation are equal, but in paralytic squint the secondary exceeds the primary. The *position of the head* affords a valuable hint as to which muscle is paralysed. Several rules are given in books, and the student can if he likes work out the direction in which a child would naturally look to compensate for the faulty position of the eye. But he will find that the patient unconsciously turns his face, or rather his chin, *towards the side of the weak or paralysed muscle.*

(3) **Diplopia,** or double vision, is the most delicate test for slight weakness or paralysis of one of the ocular muscles, even when the weakness is too slight to produce any discoverable defect in the movement or position of the eyes. To detect which is the affected muscle, hold a pencil vertically in front of the patient, and move it rapidly to the right, to the left, and in various directions, and ask him whether he can see two pencils in any of these directions. *The weakened muscle is on the same side as the direction in which the double vision appears.* But to detect

accurately which eye and which muscle is in fault further procedures may be necessary (see below). Erroneous projection, *i.e.*, error in judging the distance of objects, and vertigo (due to the same cause) are frequently associated with diplopia. The false image which is seen by the affected eye is generally hazy and less distinct than the true image seen by the sound eye. Diplopia may be homonymous or crossed. In simple or homonymous diplopia the false image lies on the same side as the affected eye: in crossed diplopia the false image lies on the side opposite to the affected eye. Paralysis of the external rectus causes homonymous diplopia, paralysis of the internal rectus causes crossed diplopia.

Method of detecting the affected eye and paralysed muscle. Place a red glass before the patient's left eye and place a candle before him in a dark room, on a level with his eyes, and about 3 yards distant. Suppose that it is found that the red image overlaps or is a little to the left of the white image, and both images are on the same level. To determine which muscle is affected, the candle must be moved to the right and to the left,

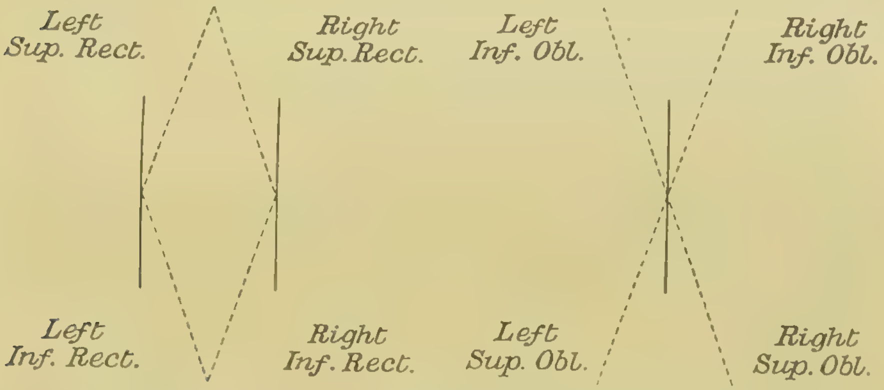

Fig. 169.—WERNER'S DIAGRAMS for detecting which is the affected muscle in cases of diplopia.

and we must notice *in which direction the distance between the images becomes increased.* Suppose that on moving it to the right the image approaches till only one candle is seen; and on moving to the left, the distance between the true and false images increases. Bearing in mind the rule that the weakened muscle is on the same side as the direction in which diplopia appears, it is evident that either the left external rectus or the right internal rectus (which turn the eyes to the left) is affected. Ask then on which side the red image appears; if on the left of the white image homonymous diplopia is present; therefore the left *external* rectus is the paralysed muscle. If, however, the red image is to the right of the white image crossed diplopia is present; therefore the right *internal* rectus is the paralysed muscle. Paralysis of the superior and inferior recti, and of the superior and inferior oblique, give rise to vertical diplopia, and there is more difficulty in discovering which muscle is affected. Loss of motion upwards is due to paralysis of the 3rd nerve; loss of motion downwards may be due to paralysis of the inferior rectus (3rd nerve) or the superior oblique (4th nerve). Werner's diagrams (Fig. 169) simplify the detection of the affected muscle in vertical diplopia. The black lines in the diagrams represent the true images, the dotted lines the false images. The dotted lines extend above and below the white lines, indicating that the false images are higher and lower than the true images. The names of the muscles in the upper and lower part of the diagrams indicate that the diplopia is caused by upward and downward movements respectively of the eyes when these muscles are affected. Thus, for example, in paralysis of the right inferior rectus an analysis of the diagram shows that (1) the diplopia occurs with downward movements of the eyes; (2) the diplopia is crossed, the false being to the left of the true image; (3) the false image has its upper part inclined towards the true image; and, (4) the false image lies lower than the true one. With the oblique muscles it must be remembered that the superior oblique moves the eye downwards, and therefore the false image due to the paralysis of the superior oblique appears on moving the eyes downwards. By remembering the diagrams it is comparatively simple to diagnose the paralysed muscle causing a diplopia.

MONOCULAR DIPLOPIA is rare. It is recognised by the persistence of the symptoms when one eye is completely closed. The symptom, which should be carefully verified, may arise in some defects of the lens, or the retina, and in some central or functional disorders.

The position and nature of the lesion in ocular paralysis is the next question to consider after having ascertained which muscle is affected in a case of squint or diplopia.

The concomitant squint of *children* in about 90 per cent. of the cases is due to hypermetropia or some other error of refraction, and when this is remedied the squint disappears. It is only rarely due to some congenital weakness of a muscle; or to a nuclear lesion like that of anterior poliomyelitis: and occasionally to intracranial lesions like those met with in adult life.

In *adults*, however, ocular paralysis assumes a much graver significance. Here we have to do with a gross or dynamic lesion involving (a) the nerve trunk of the 3rd, 4th, or 6th somewhere in front of the medulla, or (b) the nuclei of these nerves in the pons or medulla, or (c) the cortex cerebri or motor fibres passing thence to the oculo-motor nuclei.

(a) Lesion of a *nerve trunk* is the most frequent cause of squint or diplopia in adult life. In these cases we often find paralysis of a single nerve or a single muscle; there is never any conjugate paralysis (see below) with lesion of a nerve trunk. Of single muscles the external rectus is the one most commonly involved on account of the long course of the 6th nerve within the cranium; it may indeed be affected on one or both sides simply by increased intracranial pressure from a distant tumour or an acute meningitis. Other ocular nerve lesions are due to syphilitic or other neuritis, pressure by syphilitic or other growths in the orbit, pituitary fossa, or base, aneurysm of the carotid or thrombosis within the cavernous sinus (with involvement also of the 2nd and 5th nerves), or extravasation of blood after injury to the base. Acute peripheral neuritis of all the ocular nerves is rare, but sometimes is seen with alcoholic neuritis: it causes ophthalmoplegia which is only differentiated from ophthalmoplegia of nuclear origin by the absence of other head symptoms and the presence of peripheral neuritis elsewhere.

(b) *Nuclear lesions* The 3rd nerve has a long nucleus in the floor of the aqueduct. The upper or anterior end of this nucleus probably controls the pupil and accommodation, then in order the levator palpebræ, internal rectus, superior rectus, inferior oblique, and lowest of all the inferior rectus. Immediately below this comes the 4th nucleus, and below the latter the 6th nucleus, in the floor of the upper part of the fourth ventricle. Sudden and acute nuclear lesions are rare in these situations, but chronic degenerative lesions are met with. The commonest result of nuclear lesions is ophthalmoplegia externa (see below), caused by progressive involvement of all 3 nuclei; and another instance is met with in the loss of light reflex in tabes dorsalis, when the upper end of the *3rd nucleus* is affected. The simultaneous onset of paralysis of the 3rd nerve and hemiplegia (including the face) on the opposite side points to a lesion in the crus cerebri. A lesion of the *sixth nucleus* of one side, such as softening, has two peculiarities in which it differs from 6th nerve trunk lesions. (i.) Conjugate ocular paralysis results, and both eyes are deviated *away from the side of the lesion* by the unaffected muscles. This is due to the paralysis of the external rectus on the side of the lesion being associated with paralysis of the internal rectus of the other side. This is probably explained by the existence of commissural fibres connecting the two nuclei which are so frequently associated physiologically. (ii.) In some cases where the lesion is a little more extensive the entire facial nerve of the same side is also paralysed, since the fibres of this nerve twine round the 6th nucleus.

(c) *Cerebral* or *supra-nuclear lesions* never paralyse single muscles as do lesions at the base of the skull involving the oculo-motor nerve trunks. Cerebral lesions always take the form of a **conjugate deviation of the eyes and head,** the neck muscles which are so closely associated with the eye muscles in turning the head being also involved; and such lesions are attended by hemiplegia or other evidences of a cerebral lesion. There is one spot in the cerebral cortex just in front of the upper end of the Rolandic fissure which when stimulated causes conjugate deviation of the head and eyes to the opposite side. An irritative lesion in this situation, or anywhere in the course of the fibres between this and the oculo-motor nuclei will cause deviation, as already pointed out, *away from the source of irritation* (!). But destructive or paralytic lesions (which are more frequent) in this position, cause deviation *towards* the side of the lesion, from the unopposed action of the unaffected muscles. For instance, hemiplegia due to cortical hæmorrhage (an irritative lesion) is often attended during the first few days by transient conjugate deviation of the head and eyes to the opposite side. In lesions at a lower level, as in certain rare focal lesions situated in the pons,[1] the above rules as to the direction of the deviation are reversed and the deviation is apt to be of a more lasting character; destructive lesions cause a deviation of the eye or eyes and head *away* from the lesion. These are interesting examples of associated paralyses (paralyses of muscles physiologically associated) due to cerebral lesions.

(4) **Ophthalmoplegia externa** means total paralysis of all the external muscles of the eyes, the gaze being fixed and the eyes being motionless; and since partial ptosis is generally present at the same time, the patient has a sleepy expression which is very characteristic. It may occur either alone or associated with ophthalmoplegia interna (§ 605).

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[1] See a case recorded by Dr. A. Hughes Bennett and the author in *Brain*, 1889-90, vol. xii., p. 102.

Acute ophthalmoplegia is due to inflammation or hæmorrhage involving the nuclei, and is met with in diphtheria, diabetes, influenza and lead poisoning. The chronic form comes on so gradually that the patient may not have noticed it excepting by the continual necessity of moving his head to look round. It indicates a chronic degeneration of the oculo-motor nuclei, generally of syphilitic, diphtheritic, or other toxic origin. External and internal ophthalmoplegia may also occur either alone or together in some spinal lesions, such as tabes and bulbar paralysis, and in some cerebral diseases involving the cranial nuclei. Weakness of the orbicularis palpebrarum (supplied by the facial) is often met with in cases of ophthalmoplegia externa, and this is an interesting fact when we remember that the fibres of the facial nerve wind round the nucleus of the sixth.

(5) **Nystagmus** is a rapid involuntary oscillation of the eyeballs, usually from side to side (lateral nystagmus), occasionally in a vertical direction (vertical nystagmus), or in a circular direction (rotatory nystagmus). Both eyes are usually involved, though each eye should be separately examined. The movements may be constantly present, but slighter degrees can only be brought out by causing the patient to follow your finger or a bright object to the extreme left or right. If so, it indicates a weakness of the muscles on the side to which the eye is turned at that time. The symptom is notably present in disseminated sclerosis, cerebellar tumour, and Friedreich's disease and in tumour involving the corpora quadrigemina, or one side of the pons. It occurs also with meningitis, thrombosis of the sinuses and cerebral softening in various situations. It may be an indication of corneal, lenticular, retinal, or choroidal defects, especially when these start in infancy. Sometimes it is congenital, especially in albinos. It is also met with in miners, compositors, iron-founders, and those who work at close quarters, or in a strained position with deficient light.[1]

The *prognosis* and *treatment* of oculo-motor defects are those of their causal conditions. A large proportion of them are syphilitic in origin and therefore amenable to treatment. In general, recovery is more probable in nerve trunk lesions than in chronic nuclear conditions. Ophthalmoplegia interna may remain stationary for many years. For chronic cases counter-irritation and galvanism (applied through the nurse's fingers over the eyelids) may be tried.

§ **607.** VI. **Ophthalmoscopic changes** in the fundi oculorum. The **ophthalmoscope** is an instrument which is indispensable for the discovery of the affections of the retina, choroid, and optic nerve. It is used for three procedures—indirect ophthalmoscopy, direct ophthalmoscopy, and (as mentioned in § 604) retinoscopy.

The ophthalmoscope has two concave mirrors, the larger one for indirect ophthalmoscopy and retinoscopy, and a smaller one which is tilted and capable of being turned on its own axis, for direct ophthalmoscopy. Behind each of these is a hole through which the observer looks, and across which a series of lenses can be made to turn, of different refractile power. The room should be darkened, and a bright, steady light placed just beside and behind the patient's head, on a level with his ear, on the side of the eye to be examined. An Argand burner is the best, either gas or oil ; an electric lamp is not so good, as the edge of the light is not straight. Take the ophthalmoscope in your right hand and correct your own error of refraction (if any) by rotating the appropriate lens into the hole behind the mirror, through which you will look. It makes it easier for a beginner even with normal sight, to use a + 1 lens. If the patient is sitting in a chair, make him sit up (not backwards), with his head slightly forwards, about 18 inches or two feet away from your own. First learn to reflect the light steadily into his pupil, previously dilated with a 2 per cent. solution of atropine or homatropin. Tell the patient to look away into the distance over your shoulder.

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[1] Snell, *British Medical Journal*, vol. i., 1896, p. 1503.

The next step for *indirect ophthalmoscopy* is to interpose between your eye and that of the patient the convex lens. This lens should be of 2½ inches focal length, and should measure at least 2½ inches across. This lens should be held about two inches from the patient's cornea, between the thumb and index of your left hand, while the little finger rests upon the patient's forehead. You can only find the correct position of the lens by moving it backwards and forwards until a clear image of some retinal vessel is obtained. *The image of the retina seen is an inverted one.*

There are three parts to be examined ; (i.) the optic disc, which is seen by telling the patient to look in the direction of the tip of your left ear for his left eye, and *vice versâ.* (ii.) The peripheral parts of the retina may be examined by the patient turning the eye as far as possible in various directions. (iii.) The macular region is situated two disc breadths to the outer side of the disc, and is brought into view when the patient looks at the hole in the ophthalmoscopic mirror, or slightly to the side of it when the light is too strong for him to bear.

Direct ophthalmoscopy is conducted with the patient placed as before, but your head is moved up *quite close* to the patient's eye so as to look straight into it. Change the mirror on the opthalmoscope for the smaller tilted one spoken of above and rotate it on its own axis, so that its prominent *forward edge will be next to the bridge of the patient's nose* ; the lamp being on the patient's right hand for his right eye, on his left hand for his left eye, and always at the level of his ear. First adjust the ophthalmoscope accurately to your own eye (right for examining the patient's right eye and *vice versâ*) and get the handle flat against your cheek. Then tilt your head a trifle sideways close to that of the patient and tilt his head slightly the other way to avoid his breath. If the fundus is not illuminated, shift the lamp or the angle of the mirror, or your head until the red glow from the retina is seen through the little hole in the mirror. Now, to get a good image, *you must completely relax your own accommodation.* Herein lies the difficulty, but if you imagine that you are looking away at some distant object, you will by degrees acquire this necessary condition. *The image, in direct ophthalmoscopy, is always an erect one and magnified*, and therefore the different parts of the fundus can be more carefully investigated in detail than by the indirect method. To see the disc you should look obliquely inwards when the patient is looking straight in front of him, and the other parts of the retina may readily be examined by the patient moving the eyes slightly in different directions.

The *optic disc* should be examined as to its shape, its borders, its colour, its vessels, and its level. Normally the disc is slightly oval or circular, with a clearly defined border especially at the outer edge. It appears oval in astigmatic eyes. The *colour* of the disc is a rosy vermilion, but paler than the rest of the fundus. The vessels curve from the centre and then lie flat. Arteries and veins go together, but the arteries are narrower ($\frac{2}{3}$) than the veins, a trifle paler, and have a broader, more continuous light stripe running along the centre. Normally the arteries do not pulsate, but the veins, curiously enough, may do so; pulsation in the arteries may indicate (i.) increased intraocular tension, or (ii.) aortic regurgitation. The *level* is important, but a little difficult to gauge. Cupping of the disc is physiological in high myopia, and if the cup be deep the disc is always pale, a condition which should not be confused with optic atrophy. The level of the disc is best detected by the direct method of ophthalmoscopic examination. If, when thus using the ophthalmoscope, the *retina* can be seen clearly without the aid of any lens placed in the mirror hole, but the *disc* cannot be seen clearly without the aid of the lens it must be at a different level. If a little + glass is necessary to see the disc under these circumstances, then the disc must clearly be behind the retinal level (cupping). If, on the other hand, a little — lens is necessary, then the disc is on a level anterior to the retina (swelling). One can even gauge the amount of swelling or cupping in this way for, roughly, each 3 D. = 1 mm. of swelling or cupping. Thus, supposing it is necessary to use 1·5 D. to focus the retinal vessels precisely, and 4 D. to focus the disc, then there must be 2·5 D. swelling or cupping. This is an accurate method of measuring, provided the observer is able to *thoroughly relax his own accommodation.*

Two important **morbid changes in the optic disc** are met with in addition to hyperæmia and anæmia), optic neuritis and optic atrophy. In *hyperæmia*, the colour of the disc resembles the rest of the fundus. Active hyperæmia may be present in hypermetropia; passive hyperæmia is present in optic neuritis, and in the venous congestion secondary to cardiac or pulmonary disease. *Anæmia* of the optic disc is indistinguishable from an early stage of atrophy. It requires some experience to detect the difference in these delicate shades.

Optic Neuritis or Papillitis is inflammation of the optic nerve at its entrance into the globe, and is evidenced, in its typical marked form, by swelling, increased redness and vascularity of the disc, and blurring of the edges. The arteries became narrower, and the veins are enlarged and tortuous, the vessels appearing to curve over the œdematous edge. The arteries, moreover, may appear broken here and there as they are hidden by the œdema. In the early stage the disc has simply a fluffy look, and then the upper and lower edges only are blurred, a condition which is only apparent when the direct method is employed. These inflammatory changes may gradually subside, but more commonly they go on to "consecutive" atrophy. It should be remembered that the *power of vision may be undisturbed*, even when there is considerable papillitis, though the visual field is usually diminished in some degree. Disturbance of vision is generally more marked as the acute stage subsides. Early and progressive loss of vision points to damage of or pressure on the chiasma, and in such a case the pupil is dilated and immobile to light. In optic neuritis with preservation of vision the pupils react to light. *Double optic neuritis* is very strongly suggestive of intracranial disease, and especially (i.) cerebral tumour, in which it is present at some time in about 80 per cent. of the cases. It is less common in cerebral abscess and in cerebellar and other diseases in the posterior fossa, and is rare in cerebral hæmorrhage and embolism. (ii.) Increase of pressure from any other cause, *e.g.*, tuberculous meningitis, though it may be at a late stage; it is not common with simple acute meningitis. It is occasionally present in the cerebral form of disseminated sclerosis. Syphilis may produce papillitis in several ways. Various *toxic conditions* of the blood may produce optic neuritis, chief among which is renal disease, giving rise to a special form (see below). Plumbism and anæmia are occasional causes, also influenza, rheumatism, typhoid and scarlet fevers. Sudden suppression of the menses has been known to cause optic neuritis.

Unilateral papillitis indicates disease at the back of the orbit, *e.g.*, tumour or gummatous infiltration, cellulitis or periostitis.

Optic atrophy, that is, atrophy of the optic disc, is characterised by excessive pallor of the disc, a very sharply defined outline, and smallness of the vessels. In marked cases sight is completely lost, excepting, perhaps, for light. Optic atrophy may be "*consecutive*" to a severe papillitis; it may also succeed embolism of the central artery, retinitis pigmentosa, syphilitic choroido-retinitis, or pressure or injury of some part of the optic nerve or chiasma (as when blindness of one eye follows a blow on the head). *Primary* atrophy, part of a sclerosis of the optic nerve, usually arises in connection with some spinal lesion, such as tabes or disseminated sclerosis. Primary optic atrophy without other cranial symptoms should always make us suspect tabes. It is seldom possible to differentiate primary or consecutive atrophy without a knowledge of the clinical history. Optic atrophy may supervene on toxic amblyopia.

Retinitis, inflammation of the retina, is evidenced by a smokiness or haziness and loss of transparency. It may be either diffuse or patchy. Pigmentary deposits in the retina are superficial to the vessels, and appear as a network. In the choroid, pigmentary deposits are beneath the vessels and occur for the most part in rings or blotches. Choroidal atrophy appears as pale or white patches, with rings or spots of pigment in or near them. Only a few of the chief forms of retinal and choroidal disease can be mentioned here.

ALBUMINURIC RETINITIS is really a neuro-retinitis consisting of three elements—(i.) papillitis (see above), (ii.) hæmorrhages into the retina, usually most plentiful towards the disc, and (iii.) fine white shining spots near the macula and larger ones on the retina near the disc with clearly defined margins, supposed to be due to a kind of fatty degeneration of the retinal structures. One or other of these is sometimes wanting, but in its typical form this kind of retinitis is sufficiently distinctive to diagnose renal disease without examining the urine. It may occur in any form of renal affection, but is frequently associated with granular kidney. HÆMORRHAGES into the retina and choroid are met with as dark red patches, with flame-like margins. They accompany any severe retinitis or papillitis, are met with in albuminuric retinitis, also in pernicious anæmia, leucocythæmia, pyæmia, ague, scurvy, and other purpuric conditions. Retinal hæmorrhages are sometimes met with in elderly gouty persons with degenerated arteries. EMBOLISM OF THE CENTRAL ARTERY OF THE RETINA occurs most frequently in the course of cardiac disease, especially in disease of the aortic valves; and in acute and ulcerative endocarditis. It is known by the sudden occurrence of blindness in one eye in a case of cardiac disease. On examination, the retinal vessels are found empty, and a peculiar round, cherry-red spot is seen in the macular region. The disc is pale. RETINITIS PIGMENTOSA is characterised by pigmentation in stellate patches near the periphery at first, and over the vessels; the disc is greyish yellow. Night blindness is the chief symptom, and the disease starts in childhood.

Choroiditis disseminata (usually bilateral, though sometimes limited to one eye) is an evidence of syphilis (hereditary or acquired), in which disease it may arise from 3 months to 3 years after the commencement. On examination, discrete, white, atrophic patches with irregular black edges are found scattered over the fundus, *most marked at the periphery.* ATROPHIC PATCHES IN THE CHOROID may be found in the central region in myopia, or as the result of previous choroidal hæmorrhages; or after the absorption of tubercle. TUBERCLE OF THE CHOROID appears as yellow roundish ill-defined spots one-third to half the size of the disc, situated near the disc and unaccompanied by other choroidal changes.

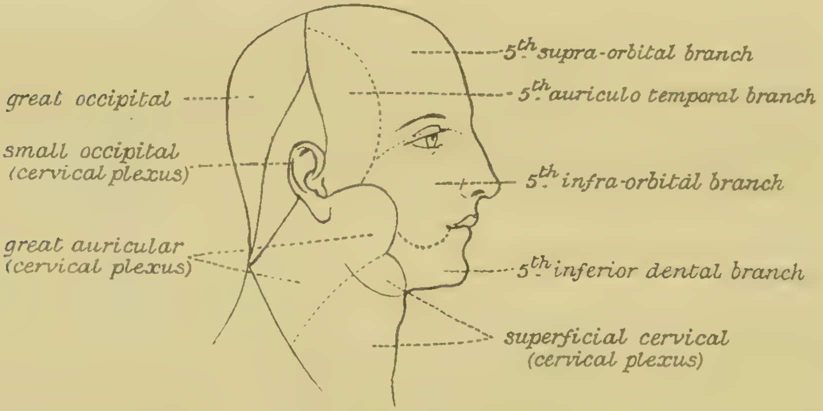
5th supra-orbital branch
great occipital
5th auriculo temporal branch
small occipital (cervical plexus)
5th infra-orbital branch
great auricular (cervical plexus)
5th inferior dental branch
superficial cervical (cervical plexus)

Fig. 170.—Nerves supplying CUTANEOUS SENSATION to the HEAD.

They may thus be distinguished from choroidal atrophy, where the spots are white, irregular, and pigmented. They appear in general tuberculosis, and in tuberculous meningitis, though usually late in the history of the case, and are therefore not of great diagnostic value.

§ **608.** The **Fifth Nerve** supplies sensation to the face, the sense of taste to the tongue, and motion to the muscles of mastication.

The fifth supplies the face and forehead, the oral, nasal, and buccal cavities, the conjunctiva and sclerotic with *sensation* (see Fig. 170); the whole of the tongue with the sense of *taste;* and it carries, as we know by the effects of lesions of its nuclei or trunk, trophic, vaso-motor and secretory (to the salivary and lachrymal glands) fibres. A *motor* root arises close beside it and accompanies its lowest or third division to supply several muscles (the temporal, masseter, 2 pterygoid, buccinator, mylo-hyoid, anterior belly of digastric, and tensor tympani). Looking upon the nuclei of the cranial nerves as continuations upwards of the anterior (motor) and posterior (sensory) grey horns of the spinal cord, the sensory portion of the fifth nerve constitutes the sensory nerve which corresponds to the facial nerve which is entirely motor.

CLINICAL INVESTIGATION. The *sensation* of the areas above mentioned can be tested in the usual way. When it is lost the patient in drinking feels only one half of the tumbler, and may imagine it is broken. The *motor* functions of the fifth are tested by asking the patient to clench his teeth while you place the fingers on the temporal muscles of both sides and then on the masseters. It may be inferred that the pterygoids of one side are paralysed if on opening the mouth or projecting the chin forward the jaw deviates towards one side, *i.e.*, towards the paralysed side (by the action of the unaffected pterygoids of the opposite side). Paralysis of the tensor tympani produces a diminution of hearing for high notes. The function of *taste* will be considered below. This should always be tested in suspected lesions of the trunk or ganglion of the 5th.

The *symptoms* of *destructive* or paralytic lesions of the fifth nerve or nucleus consist of the following alterations according to the part involved —Anæsthesia (preceded perhaps by severe burning pain) of the area supplied by the fifth; loss of conjunctival, palatal and nasal reflexes on that side; loss of secretion of lachrymal and salivary glands; loss of smell owing to the dryness of mucous membrane; loss of taste; diminished masticatory power and collection of food between cheek and jaw; hemiatrophy facialis (§ 612) occasionally.

Irritative lesions of the fifth nerve give rise to facial neuralgia and hyperæsthesia or tender points. Irritative lesions of the Gasserian ganglion are attended also by very serious trophic changes—cloudiness of the cornea going on to perforation and destructive panophthalmitis, herpes frontalis, or vesicles in other positions, and in rare cases sloughing of the mucous membrane of the cheeks. *Reflex* irritation of the fifth nerve may give rise through the facial nerve to twitchings of the face, or through the optic nerve to flashes of light.

Lesions of the fifth. (i.) Lesions of the *cerebrum* involving the sensory crossway of Charcot imply facial as well as corporeal hemianæsthesia, both of which are on the side opposite to the lesion. (ii.) Lesions *within the pons* attended with corporeal hemianæsthesia produce a "crossed" anæsthesia, face on same side, body on the other. Lesions, however, such as hæmorrhage, softening, or tumour in this situation more frequently involve part of the widespread fifth nucleus than the whole. (iii.) *Within the cranial cavity* syphilitic meningeal conditions, caries of the petrous bone on the tip of which the Gasserian ganglion rests, tumours, aneurysms, and very rarely fractures of the base, produce total or partial paralysis of the fifth. Disease in the pituitary region, cavernous sinus or orbit, *e.g.*, aneurysm of the internal carotid, or orbital cellulitis, damage the 1st division of the nerve. (iv.) *Outside the cranial cavity* one or other division may be paralysed by *tumours*—tumours of the parotid for instance, extending into the spheno-maxillary fossa, and damaging the 2nd and 3rd divisions of the nerve. The deep position of the fifth usually protects it from *injury*, and *neuritis* is said to be rare. It is, however, to my mind very difficult to account for the fact that the branches of this nerve form the favourite seat for neuralgia, and that herpes so constantly occurs, excepting on the supposition that those branches, or the nerve or its ganglion, are the seat of neuritis. The fifth nerve is a frequent seat of *reflex irritation* from distant parts (neuralgia, § 598): it is also the source of reflex irritation in cases of facial twitchings, cough, and other motor spasms.

The *prognosis* and *treatment* of neuralgia of the fifth has already been dealt with (§ 598): the prognosis and treatment of the other lesions depend upon their nature.

§ 609. The sense of **taste** should always be tested in suspected lesions of the fifth, which is the nerve of taste. Powdered salt, sugar and quinine are necessary. The patient should put out his tongue and keep it out till the end of the test. Having wiped the tongue place on it a minute portion say of powdered sugar, gently rub in, and ask "is that salt?" "is it bitter?" "is it sweet?" The patient can reply by shaking or nodding his head. Citric acid may also be employed. A weak galvanic current causes a metallic taste. Sweets and acids are tasted best by the anterior part of the tongue, bitters by the posterior part.

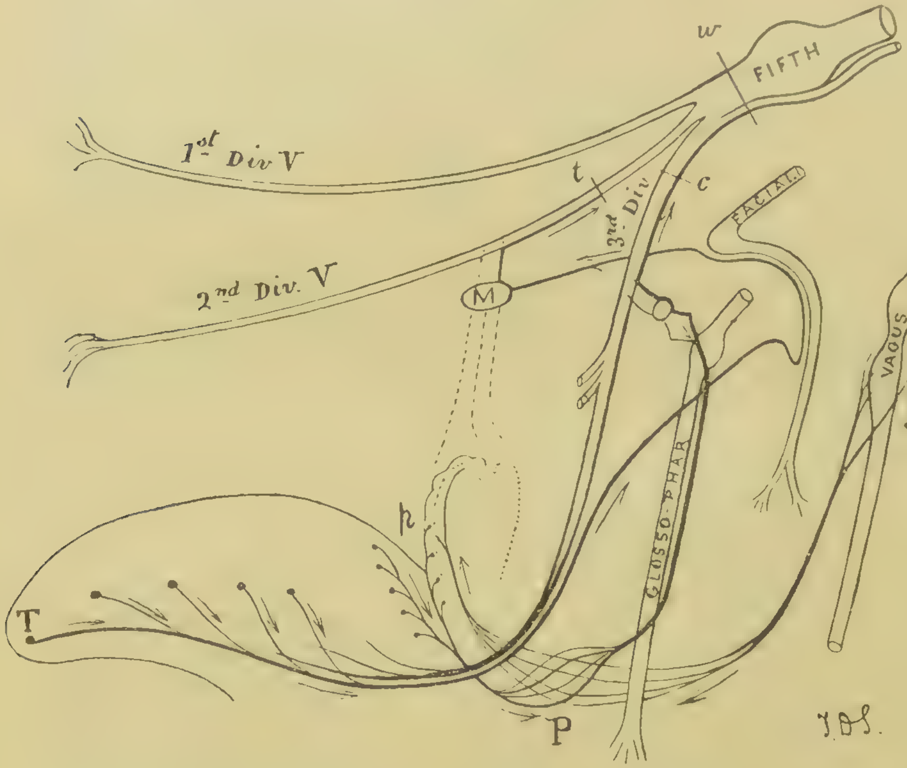

Fig. 171.—Sense of Taste and nerve supply of palate. Diagram to show how taste impressions reach the 5th nerve ⋙⟶, and the motor supply of the palate ⟵⋘. T, branches of the chorda tympani conveying taste from the tip and sides of the tongue and running with the lingual branch of the 5th, then through the facial to Meckel's ganglion (M) and thence to the 2nd division of the 5th. *p*, soft palate from which, and from the dorsum of the tongue, taste fibres pass through the pharyngeal plexus (P) to join the glosso-pharyngeal, and thence through the otic ganglion (*o*) to the 3rd division of the 5th. The palate, it will be seen, is supplied by the accessory portion of the spinal accessory through the vagus.

Tract of taste (Fig. 171). Between the tongue and the main trunk of the fifth nerve taste is conveyed by two separate and circuitous routes. (a) The chorda tympani running in the tongue with the lingual branch of the 3rd division of the fifth collects taste fibres from the anterior two-thirds of the tongue (some say the tip and anterior two-thirds of the edge). The chorda then joins the facial nerve just above its exit from the stylo-mastoid foramen, and after running its well-known course through the tympanum doubles back as the Vidian nerve from the facial to Maeckel's ganglion, and thence to the second division of the fifth. (b) From the middle and posterior part of the tongue taste fibres pass first along the glosso-pharyngeal nerve, which they leave by Jacobson's nerve to join the tympanic plexus, thence by the small superficial petrosal and the otic ganglion they pass to join the second division of the fifth. After reaching the pons the path of taste decussates and reaches the posterior part of the internal capsule of the opposite side, and is believed to terminate in the cortex at the tip of the temporo-sphenoidal lobe.

The commonest *cause* of loss of taste in the tip and edges is (1) a lesion of the facial in the fallopian aqueduct, as in middle ear disease. (2) Loss of taste in the posterior part of the tongue is met with in lesions of the third division of the fifth; of the anterior part, in lesions of the 2nd division; and of the whole of one side of the tongue in lesions of the trunk of the fifth just outside the pons. (3) Loss of taste of one half of the tongue occurs in many cases of hemianæsthesia, whether of functional or organic origin. (4) Disturbances of taste are frequent in influenza, diphtheria, and some other acute specific disorders. (5) Taste is greatly aided by the sense of smell. Flavours, such as those of cheese, wine, or roast beef, really consist of compound perceptions of smell and taste. Consequently catarrh and many nasal diseases are attended with impairment of taste.

Paragusis (perverted taste) and hyperagusis (increased sensibility to taste) are met with as auræ to epileptic fits, in conjunction with insanity, with hysteria, and sometimes with middle ear disease.

§ 610. The **Facial Nerve** (the seventh) is a purely motor nerve supplying all the muscles of the scalp and face (except the levator palpebræ superioris), the platysma, two small muscles of the hyoid bone and the stapedius. The facial and the sixth are more frequently involved singly than is any other cranial nerve. The facial is also very frequently partially involved in cases of hemiplegia.

Anatomy of the FACIAL NERVE (Fig. 172). The *nucleus* of the facial nerve is situated in the floor of the fourth ventricle, just beneath the most prominent part of the eminentia teres. Its lower end is in close proximity to the nucleus of the twelfth (hypoglossal), a fact which corresponds to the close association between the muscles of the lips and those of the tongue. There is good reason to believe that the orbicularis oris is innervated from the *nucleus* of the hypoglossal (see bulbar paralysis). Its upper end is close to the sixth (abducens) nucleus, around which its fibres curve as they pass to the superficial origin. The *cortical centre* for the face is situated at the lower end of the ascending parietal convolution, and this centre is connected with the facial nucleus *through the internal capsule*. Hence the frequency with which the lower half of the face is affected in cases of hemiplegia. The facial fibres cross to the opposite side in the upper part of the pons, and appear on the surface at the lower edge of the pons. The nerve then accompanies the eighth (auditory) nerve, to the internal auditory meatus. At the lower end of the internal auditory meatus, the facial nerve presents a swelling (the geniculate ganglion), which gives off three important branches. It then enters the aqueductus fallopii, which curves forward over the foramen ovale on the inner wall of the tympanum, then passes downwards and out through the

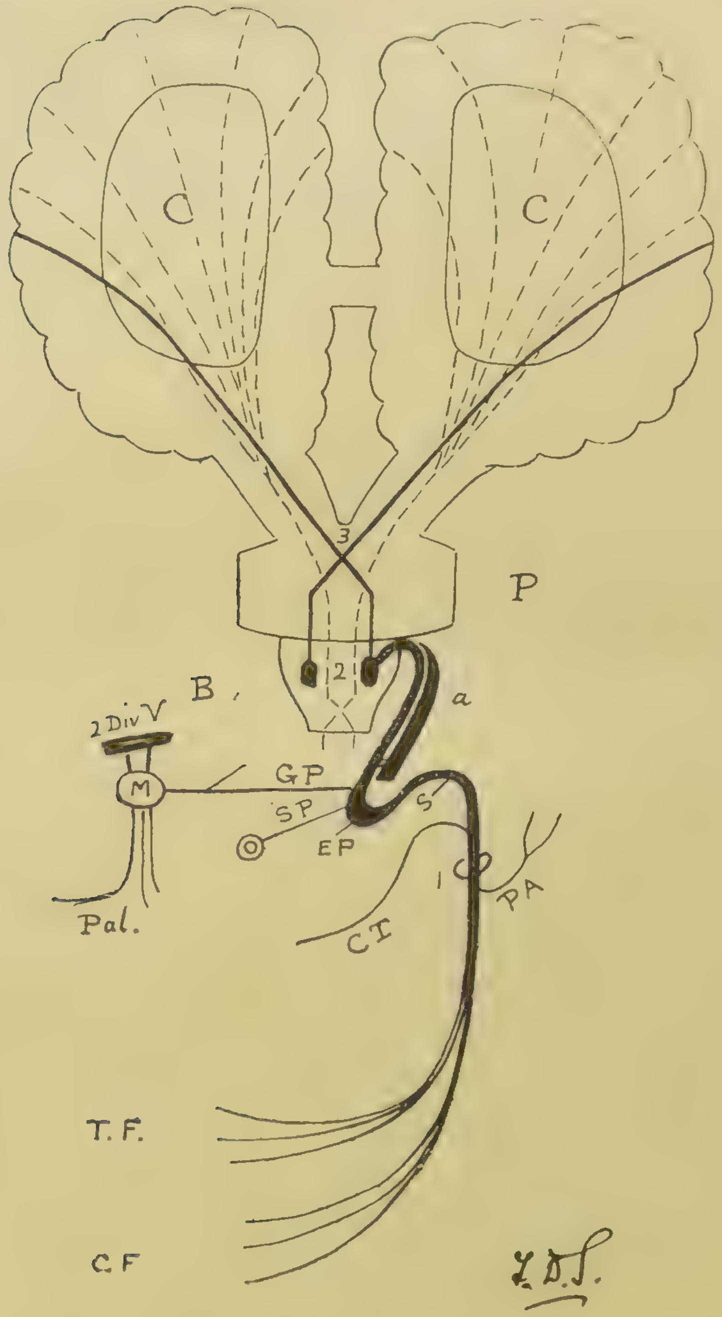

Fig. 172.—The Facial Nerve, its course and connections. 1, stylo-mastoid foramen; 2, above pyramidal decussation; 3, decussation of facial fibres in the pons. C.T., chorda tympani; S., branch to the stapedius; E.P., S.P., and G.P., external, superficial, and great petrosal nerves; O., the otic, and M., Meckel's ganglia; P.A., posterior auricular branch; *a*, auditory nerve; Pal., sensory branches from the palate to the 2nd division of the 5th (compare fig. 171).

stylоid foramen, and while traversing the parotid gland breaks into its two main terminal divisions, the temporo-facial to the muscles of the upper half of the face, the cervico-facial to those of the lower half, the platysma, the mylo-hyoid, and the posterior belly of the digastric.

The first and clinically most important branch of the facial is the great petrosal (or Vidian) nerve, which joins the geniculate ganglion to Maeckel's ganglion. Maeckel's or the spheno-palatine ganglion is connected above with the second division of the fifth, and the lower branches coming off from it supply the palate (Fig. 171). It was formerly thought that these were motor branches originating from the facial, but clinical research shows that the great petrosal nerve is really sensory, and contains taste fibres derived from the second division of the fifth, which join the geniculate ganglion, pass along the facial trunk, and leave the facial trunk as the chorda tympani nerve. It was Hughlings Jackson who first declared he had never seen undoubted paralysis of the palate in association with lesions limited to the facial nerve, that the palate is to a certain extent asymmetrical in most people, and that this fact had not hitherto been sufficiently allowed for. It is, moreover, a clinical fact which Gowers has insisted upon, that loss of taste is only associated with lesions of the facial nerve situated at some point between the geniculate ganglion and the styloid foramen. Lesions of the facial nerve behind the latter point do not give rise to loss of taste. Clinically, it is of great importance to remember that the fallopian aqueduct is pierced by three structures, and through these holes inflammation may spread to the facial nerve. The first contains a small nerve twig from the facial nerve to the stapedius muscle. The second contains an arterial twig from the tympanum for the nutriment of the structures in

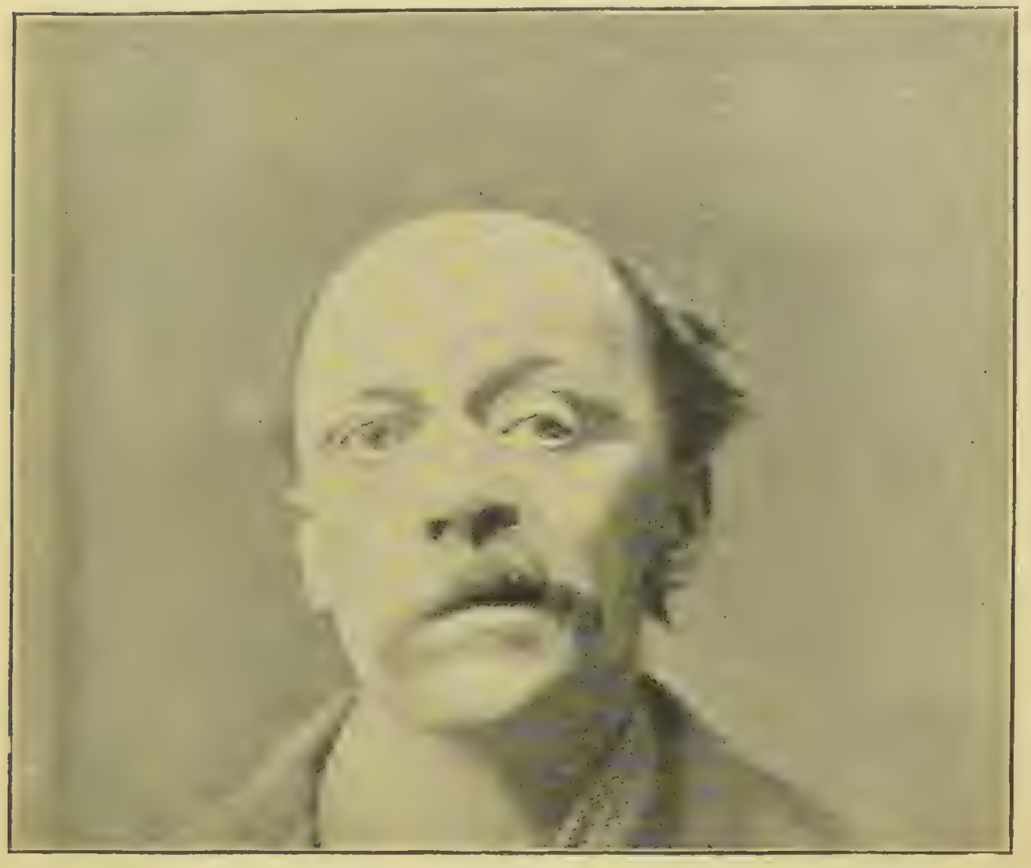

Fig. 173. -FACIAL PARALYSIS. This picture appears to represent *right* facial paralysis, but in reality it represents the face of a man aged 42 with paralysis of the *left* side, which had occurred 2 years previously consequent on middle ear disease. It exhibits, therefore, late contracture of the paralysed muscles on the left side.

the aqueduct. The third contains the chorda tympani nerve which comes off from the facial a quarter of an inch above the stylo-mastoid foramen, and passes forwards between the handle of the malleus and the stapes, turns downwards, and after giving a vaso-constrictor branch to the submaxillary ganglion, terminates in the tongue, which it supplies with taste fibres at the tip and along the anterior two-thirds of the margin.

In the CLINICAL INVESTIGATION of cases of facial paralysis, as in other disorders of the nervous system, the first step is to discover the position of the lesion and then its nature. This is done by investigating, first, the muscles that are affected; secondly, the condition of the hearing; thirdly, the condition of the taste; and fourthly, the electrical reactions.

The *symptoms* of complete paralysis of the facial nerve (Bell's paralysis) are (i.) an obvious one-sided alteration of the face which is pulled up away from the paralysed side by the unaffected muscles, the forehead and cheek of the paralysed side being smooth and expressionless. (ii.) The patient cannot shut his eyes, or in slight cases he cannot keep them shut when we try to forcibly open them. (iii.) The corner of the mouth is drawn up when he attempts to screw up the eyes, he cannot smile or show his teeth. He cannot whistle, and the food collects between the cheek and teeth. (iv.) The taste and (v.) the hearing may be affected in certain cases (see below). Paresis of the palate used to be mentioned, but the palate is now known to be supplied by the spinal accessory. In the course of many months the *affected side becomes drawn up* by contraction of the paralysed muscles (Fig. 173). Facial paralysis must not be confused with hemiatrophy facialis (§ 612).

The **varieties of facial paralysis** differ according to whether the lesion is (a) in the brain, (b) in the facial nucleus, or (c) the facial nerve. (a) is incomplete, but (b) and (c) are complete (or Bell's) paralysis. What follows should be compared with Fig. 172.

(a) With CEREBRAL (SUPRA-NUCLEAR) LESIONS facial paralysis is extremely common, and it differs considerably from that of lesions situated at or below the facial nucleus. (i.) It is usually associated with hemiplegia on the same, or occasionally on the opposite side. If (as is usual) the hemiplegia is on the same side as the facial palsy the lesion is situated in the internal capsule or above the pons, but in crossed hemiplegia it is situated *in* the pons. (ii.) The upper half of the face is exempt from the paralysis (the patient can close his eyes), because, it is suggested, a certain number of the fibres of the facial nerve escape destruction. Intra-nuclear lesions, on the other hand, give rise to paralysis of all the facial muscles, Bell's paralysis as it is called. (iii.) The muscular power is speedily restored; and (iv.) there are no electrical changes. The anatomical lesions are those of hemiplegia (*q.v.*). Hysterical facial palsy is extremely rare; it is slight and the platysma is said to be exempt.

(b) NUCLEAR LESIONS are rare. (i.) Atrophy and marked R. D. supervene early. (ii.) Diplopia may be present owing to the proximity and involvement of the 6th nucleus. (iii.) All the facial muscles are affected, but some more than others. If the lips are mainly affected, we must suspect glosso-labio-laryngeal paralysis, and should investigate movements of the tongue and larynx. If the lips are exempt, and the upper part of the face alone is paralysed, we have probably to do with a nuclear lesion affecting chiefly the upper part of the nucleus, such as arises in rare cases of diphtheria, spinal affections such as tabes and disseminated sclerosis, rare cases of focal lesion, and anterior poliomyelitis spreading upwards.

(c) INFRA-NUCLEAR LESIONS give rise to paralysis of *all the facial muscles* on one side (**Bell's paralysis**), but the other symptoms will vary somewhat with the precise locality of the lesion.

(1) Lesions of the *facial trunk* just in front of the pons are known by (i.) complete paralysis of all muscles; (ii.) R. D. and wasting; (iii.) possibly some nerve deafness by involvement of the adjacent auditory nerve; but (iv.) taste is always normal. Gummatous and other tumours may affect the nerve here.

(2) Lesions in the *fallopian aqueduct* (perhaps the commonest position) are known by (i.) complete paralysis of all the muscles, coming on very suddenly; (ii.) R. D. and wasting in the course of a week or two; and (iii.) taste of tip and side of the tongue lost, owing to paralysis of the chorda tympani, this being the pathognomonic feature of neuritis or other lesions in the aqueduct. (iv.) There is no nerve deafness, but the hearing is generally affected. One of the commonest causes of facial paralysis is disease of the middle ear, the inflammation spreading to the facial nerve in the aqueduct, a condition of some gravity because the bony walls do not allow of the expansion of the nerve, and the compression aggravates the condition. In some cases paralysis of the stapedius muscle may, in the absence of otitis media, give rise to increased acuteness of hearing, especially for low tones, on account of the unopposed action of the tensor tympani.

(3) *Neuritis external to the skull* (rheumatic or other) produces (i.) paresis of all muscles, but usually incomplete; (ii.) recovery usually ensues in a short time; (iii.) there may be some diminution of faradic reaction, but no R. D., and (iv.) no loss of taste unless the inflammation spreads into the aqueduct.

Among the *causes of Bell's paralysis* otitis media or some other affection of the middle ear is perhaps one of the commonest, as above mentioned, and a history or evidence of this is often obtainable. A simple sore throat may spread to the middle ear and cause facial paralysis. Exposure to chill, as for instance in a railway carriage, is certainly a very frequent cause of facial paralysis, especially of variety c (3). It sets up a neuritis or peri-neuritis, which may spread up the nerve into the aqueduct, as evidenced by an associated loss of taste in some cases. It occurs for the most part in gouty or rheumatic people, and those between 20 and 35 years of age, of either sex. In nearly three-fourths of 19 cases of facial paralysis lately under my care a history of chill or exposure could be revealed. The paralysis comes on quite suddenly, in the course of 24 to 48 hours after the exposure, and is rarely quite complete. In the course of three days there may be some loss to faradism, but in the course of 4 or 5 weeks the paralysis generally clears up. Fracture of the base may result in facial paralysis, forceps applied during birth, or boxing the ears in childhood, as in a case mentioned by Gowers. Basal meningitis, especially of syphilitic origin, may involve the facial nerve, but usually some other cranial nerve as well. Rare cases of haemorrhage into the aqueduct have been confirmed by post-mortem. Inflammation and new growths of the parotid are generally sufficiently obvious as causes of the condition. Diphtheria and alcohol are occasional causes.

The *prognosis* of facial paralysis as to recovery depends partly upon the position and partly upon the cause of the lesion. The prognosis of supranuclear conditions depends upon the concomitant symptoms. The prognosis is much more serious in all lesions within the skull than those external, and especially when the lesion is within the aqueduct, which invariably results in permanent degeneration of the nerve. In cases of external neuritis due to chill, recovery is the rule. The electrical changes give us valuable information as to prognosis. My general rule is as follows: —If there are no or very slight electrical changes in the second or third week, recovery is the rule, but if the reaction to faradism remains markedly lowered, and especially if it is quite lost for three months, the chance of recovery is small, if for six months, *nil.*

The *treatment* in most cases is hopeful. It must be directed to the cause, and the assistance of an aural surgeon is generally required. Even apart from syphilis, iodide of potassium in small doses is very useful in rheumatic cases, especially if combined with quinine. As a remedial measure galvanism is of great use to restore the nutrition of the muscles and the function of the nerve, if applied regularly.

§ 611. **Diplegia facialis,** or double facial paralysis, is somewhat difficult of detection, but it is characterised by a total absence of expression, an inability to close both the eyes, and an absence of adequate movement around the mouth in talking. It is a much more serious condition than one-sided facial paralysis because of the lesions which it suggests.

Double facial paralysis may be *caused* by some of the lesions previously mentioned occurring on both sides, such as (i.) basal lesions (*e.g.*, syphilitic meningitis or tumours), or (ii.) double otitis media. In both of these it generally happens that first one facial nerve is affected, and then some weeks or months later the nerve of the other side., (iii.) Diphtheria, and (iv.) focal lesions in the upper part of the pons may affect both sides at once. Diphtheria is, I believe, a more frequent cause of slight double facial paralysis than is generally recognised; it is evidenced by the expressionless aspect of children suffering from diphtheritic paralysis. (v.) Focal lesions in the upper part of the pons in the position of the decussation of the facial fibres are extremely rare.

§ 612. **Hemiatrophy facialis** is a rare condition consisting of atrophy of the skin and its appendages, the subcutaneous tissue, and sometimes of the bones on one side of the face, unattended by alterations in the sensation or muscles of the face. It must not be confused with facial paralysis. It is believed to be due to a degenerative lesion (possibly of the nature of poliomyelitis) of the upper part of the nucleus of the *fifth nerve*, with atrophy of the upper part of the nerve trunk within the pons. One case observed by the author[1] followed a severe neuralgia which dated from an injury to the vertex.

§ 613. The **auditory nerve** (the eighth) is the nerve of hearing and orientation.

It arises by 2 roots. The dorsal or auditory root arises from the internal auditory nucleus and passes out behind the restiform body. The ventral root (orientation) arises from the external auditory nucleus and passes out in front of the restiform body. The two roots join and enter the internal auditory meatus to be distributed, the former to the cochlea, the latter to the semicircular canals. The cortical centre for hearing is in the 1st and 2nd temporo-sphenoidal convolutions of the opposite side; that for orientation is unknown. The auditory apparatus is shown in Fig. 174.

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[1] Trans. Clin. Soc. Lond., 1903.

CLINICAL INVESTIGATION. *Hearing* should be tested by the voice or a watch, and a tuning-fork. The presence of wax in the external meatus must be first excluded, and if necessary the ear should be syringed with warm water after the wax has been softened by warm oil or bicarbonate of soda solution (2 teaspoonfuls to the pint). To test the *acuteness* of *hearing*, stand behind your patient, close one of his ears with one of your hands, and place a watch in the other hand outside the range of his hearing, then approximate it slowly, asking the patient to speak directly

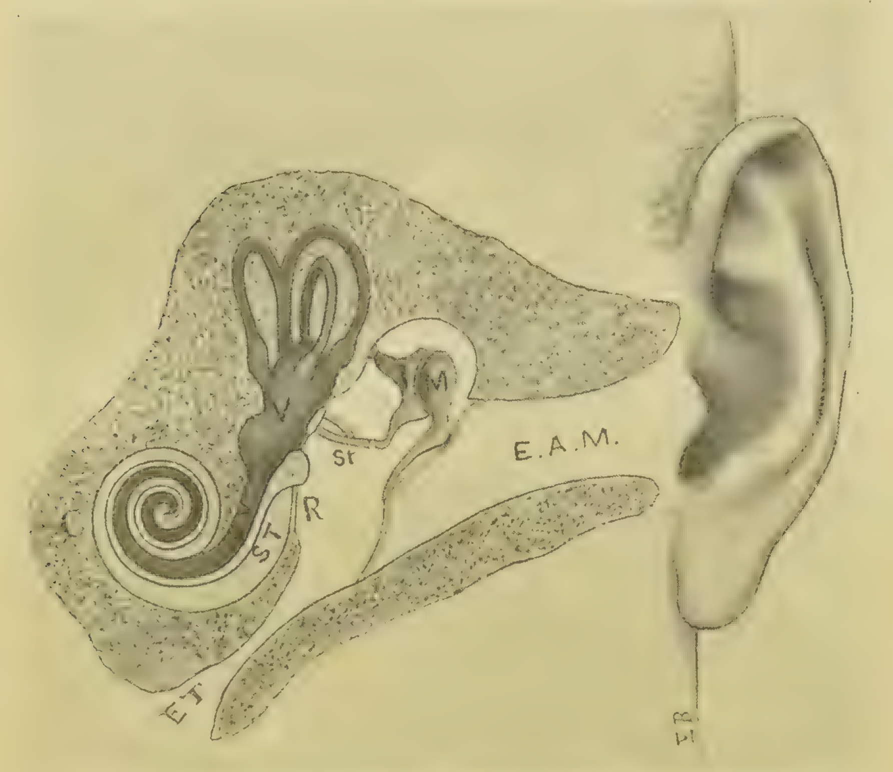

Fig. 174.—AUDITORY APPARATUS (diagrammatic representation) of the Left side seen from the front, the internal parts being magnified two-fold.

E.A.M., External auditory meatus, separated by tympanic membrane from tympanum in which R. is situated.
M., on head of Malleus.
I., Incus fixed to wall by its short process and articulating with the stapes by its long process.
St., Stapes, the foot of which fits into the fenestra ovale.
V., Vestibule consisting of saccule (below) and utricle (above). Into the latter open the 3 semi-circular canals, Superior, Posterior, and External (or Horizontal). The vestibule leads on to the Scala vestibuli (S.V.) of the cochlea (C.).
R., Fenestra Rotunda leading from the Scala tympani (S.T.) to tympanum.
E.T., Eustachian tube.

he hears the tick, and then estimate the distance. Examine the other ear in the same way. Ascertain on yourself what is the normal distance at which that particular watch should be heard, and supposing this is 60 inches, and the patient hears with the left ear at a distance of 5 inches, and with the right at 60 inches, then the acuteness of his hearing is represented by the fraction $^{5}{}_{60}{}^{60}$. The hearing for the voice is indicated by the distance at which the patient can hear whispered sounds; or by noting whether at 2 yards the patient can hear whispered, ordinary, loud, or shouting conversation.

Weber's test. To ascertain whether an impairment of hearing is due to *nerve deafness* or to *obstructive deafness*, test the perosseus hearing by placing a watch or a vibrating tuning-fork on the mastoid bone or on the centre of the patient's forehead. If the deafness is due to disease of the auditory nerve it cannot of course be heard in the affected ear, or at any rate not as well as in the good ear. This is known as Weber's test Negative, and indicates Nerve disease. If the deafness is due to obstructive ear disease (the nerve being intact) the sound will be heard quite as well and probably *better on the defective side* than on the healthy side (Weber's test positive).

Rinné's test. In a normal person a tuning-fork placed on the mastoid bone until no longer heard in that situation by the patient, can still be heard by him if moved and held opposite the meatus (Rinné's test positive); it indicates an absence of middle ear disease. When the middle ear or conducting apparatus is definitely diseased the tuning-fork cannot be heard opposite the meatus after it has ceased to be heard when held on the mastoid (Rinné's Negative).

Galton's whistle is for testing the upward limit of audition of a patient. Diminution of audition for high-pitched notes occurs in old age and in incipient nerve deafness. *Paracusis Willisii*, "hearing better in a noise," is a characteristic of bilateral middle ear disease. Such patients can hear conversation better in a train or omnibus. In boiler makers' and some other forms of nerve deafness the converse is true.

INSPECTION OF THE EAR. Note should be made of any discharge and its character (see below), any pain or tenderness over the mastoid (see below), any eczema of the meatus, etc. To examine the *meatus* (which should be done first without a speculum) the auricle should be pulled gently upwards and backwards, the tragus being held forwards by a blunt probe. If a speculum is to be used the auricle should be held between the middle and ring fingers of your left hand (for the patient's right ear), the speculum being inserted with your right hand inwards and slightly downwards and forwards. The speculum can then be held between the thumb and forefinger of your left hand. It facilitates examination to have a mirror on the forehead as in laryngoscopy (§ 121) to reflect the light from the side of the patient's head. The convexity of the floor of the meatus may be mistaken for an abscess or polypus. Cerumen is of dark colour and soft consistence. The membrana tympani may present indrawing (due to blocking of the Eustachian tube), congestion, thickening or loss of lustre, atrophic areas, or perforations.

The NASO-PHARYNX should be next examined. Note (1) the activity of the palatal muscles, (2) the Eustachian tubes and back of the nose, by posterior rhinoscopy, and (3) the patency of each nostril.

The PATENCY OF THE EUSTACHIAN TUBE is usually tested by inflation of the middle ear by *Politzer's* method. The nozzle of the rubber bottle is inserted into one nostril, and both nostrils are then held closed between the thumb and finger of the operator. The patient is then directed to swallow or to say "hic," and at the same moment the air from the rubber bottle is forced into the nose. Deglutition raises the palate and opens the Eustachian tube, and the air having no other outlet is forced into it. A tube connecting the ear of the patient with that of the operator will enable the latter to hear an audible "pick" if the middle ear is inflated, and this will reveal the patency of the Eustachian. A second point to note is the effect which inflation has upon the symptoms—the hearing, tinnitus, or pain. The hearing is temporarily improved in middle ear or Eustachian disease, made worse in nerve deafness. In *Valsalva's* method of inflation the patient pinches his nostrils firmly and makes an expiration as if to blow his nose, but without allowing the air to issue. The *Eustachian catheter* is sometimes required to inflate for diagnosis or treatment. It is not a difficult operation, but requires a little practice. Pass it tip downwards very gently along the floor of the nose to the edge of the hard palate, the patient being directed to breathe through the nose so that the soft palate may droop. Immediately the tip of the catheter has reached the edge of the hard palate turn it upwards and outwards, and it will enter the Eustachian orifice; it may be aided by the patient swallowing at the same time. The nozzle of Politzer's bag may now be carefully introduced and inflation performed as before.

Two kinds of deafness are recognised by aural surgeons; **nerve deafness** due to lesions of the auditory nerve, and **obstructive deafness** due to some disease in the middle ear or auditory passages.

DIAGNOSIS OF NERVE DEAFNESS AND OBSTRUCTIVE DEAFNESS.

Nerve deafness.	*Obstructive deafness.*
Diminution of bone conduction.	Loss of air conduction only, with negative Rinné.
Loss of hearing for very high pitched tones.	Better hearing for very high than for very low tones.
Decreased hearing in midst of noise.	Increased hearing in midst of noise.
Hearing for conversation relatively better than for watch.	Hearing for conversation relatively worse than for watch.
Tinnitus occasional.	Tinnitus usual.

§ **614.** The **causes** of **deafness** slightly altered from Dr. Dundas Grant's arrangement[1] may be summarised as follows:—

Nerve deafness:—*a. Coming on* GRADUALLY WITHOUT EVIDENCES OF INTRACRANIAL DISEASE *may be due to*:—

1. Toxic causes (*e.g.*, quinine, salicin, tobacco).
2. Chronic anæmia, or congestion of the labyrinth.
3. Paresis from chronic concussion—machinery, guns, etc.
4. Hysterical deafness.

b. Deafness coming on GRADUALLY WITH INVOLVEMENT *of other* NERVOUS STRUCTURES.

1. With facial paralysis, would indicate either disease of the petrous

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[1] *Medical Annual*, 1895.

bone (when there would be discharge of some kind) or gumma, tumour, or pachymeningitis within the skull.

2. With the 6th nerve or spinal accessory—basal pachymeningitis, or a tumour.

3. With hemiplegia of the same side—disease of the temporo-sphenoidal lobe or motor strands below.

4. With crossed hemiplegia—disease of the pons.

5. With hemianæsthesia—disease of the hinder part of the interior capsule.

6. With failure to understand spoken but not written words, without loss of speech (word deafness)—disease of the left superior temporo-sphenoidal lobe.

7. With locomotor ataxy or disseminated sclerosis—degeneration of the auditory nucleus.

c. Nerve deafness of SUDDEN ONSET.

1. With sudden vertigo, vomiting, etc., would indicate Ménière's disease (§ 524a).

2. With sudden hemianæsthesia or hemiplegia—hæmorrhage into the temporo-sphenoidal lobe, internal capsule or pons.

3. If dating from a mental or emotional shock—neurotic or hysterical deafness.

4. If dating from forcible syringing, loud noises, explosions, or blows on the head—concussion of the labyrinth. Fracture of the base may be attended by fracture of the capsule of the labyrinth.

d. Nerve deafness of MODERATELY ACUTE ONSET.

1. Without pyrexia, nerve deafness may come on within a few hours in syphilitic disease of the labyrinth. Acute congestion and acute anæmia of the labyrinth coming on with nausea and giddiness are also causes.

2. With pyrexia, primary inflammation of the labyrinth may supervene with cerebral symptoms during or after various specific fevers, meningeal inflammation of the auditory nerve, or injury.

Obstructive deafness, or deafness due to disease of the middle ear or auditory passages rarely comes on (a) SUDDENLY, excepting from impaction of cerumen. If it comes on (b) ACUTELY, it is probably due to acute catarrh of the Eustachian tube.

CHRONIC OBSTRUCTIVE DEAFNESS OF SOME STANDING:—

a. Without a history of previous discharge.

1. If the deafness dated from an acute naso-pharyngeal catarrh, tinnitus is not constant, inflation with Politzer's bag gives some relief, and on inspection the tympanic membrane is indrawn, opaque, and thickened, the disease is the exudative form of chronic catarrh of the middle ear.

2. If the deafness had an insidious onset, tinnitus is a prominent symptom, inflation gives no relief, and on inspection the tympanic membrane is somewhat opaque, in parts thinned and in other parts thickened, the disease is the sclerotic form of chronic catarrh of the middle ear.

b. With a history of previous purulent discharge. The deafness is probably due to perforation or cicatrices resulting from suppurative inflammation of the middle ear, and inspection of the drum confirms this.

Combined Obstructive and **Nerve Deafness.** In this condition it is sometimes a little difficult to make out the exact state of matters. However, such cases may be grouped into those with and those without discharge.

a. If there is a *history or presence of discharge*, the commonest condition which gives rise to it is a suppurative otitis media spreading to the labyrinth. In these circumstances we get signs of nerve deafness gradually supervening on those of obstructive deafness.

b. If there be *no discharge, past or present*, the most usual conditions are:—1. Disease of the cochlea or ankylosis of the stapes supervening on an old chronic catarrh of the middle ear. 2. If the history of nerve deafness precedes the obstructive deafness, the middle ear catarrh has supervened on the nerve deafness.

Pain in the ear may be due to:—1. Otalgia, when there is no sign of local disease or defective hearing, and a reflex cause such as a bad tooth is present. 2. Disease of the external meatus, such as furuncle, or eczema. 3. Disease of the middle ear, when there is deafness, some pyrexia, and examination reveals congestion of the membrane.

PAIN IN THE MASTOID REGION may be due to:—1. Mastoid neuralgia, which sometimes follows old mastoid disease. 2. Accompanied by redness, swelling and tenderness—inflammation of the mastoid lymphatic gland, periostitis or abscess. 3. Accompanied by deep throbbing, pain and constitutional disturbance, it may be due to internal mastoiditis, which is often consequent on chronic suppuration.

Pain more or less GENERALISED OVER THE HEAD, accompanied by PYREXIA, may be associated with the following diseases of the ear:—

a. Acute diseases:—1. Acute middle ear suppuration, which is relieved by outlet of pus; 2. mastoiditis; 3. acute meningitis; 4. pyrexia.

b. If associated with a *history of chronic suppuration* from the ear:—1. If the temperature is continuously high, it may be due to retention of pus, extradural abscess, or meningitis (§ 542). 2. If the temperature oscillate, there may be pyæmia or sinus thrombosis (§ 544). 3. If the temperature after an initial rise is normal or sub-normal, and there are headache, slow pulse and delayed cerebration, suspect abscess of the temporo-sphenoidal lobe (§ 543).

Discharge from the ear. A STICKY OOZING discharge may be due to eczema of the meatus or condylomata. A HÆMORRHAGIC discharge may be due to vascular granulations or erosion of blood vessels occurring with middle ear disease, or (rarely) vicarious menstruation in hysteria. An offensive SANIOUS discharge with fungating granulations, acute radiating neuralgia, and enlargement of the neighbouring glands is characteristic of malignant disease of the ear.

A PURULENT discharge (a) *which is or has been copious*, and associated with deafness from the beginning of the symptoms, is due to acute or chronic suppuration of the middle ear. When associated with chronic suppuration it may be due to the presence of polypus, granulations or cholesteatoma, caries of the malleus, incus or temporal bone, disease of the mastoid antrum or naso-pharynx, or to constitutional causes such as diabetes mellitus, tubercle, anæmia or syphilis.

(b) A PURULENT discharge *which is not and never has been copious*, and deafness if present did not supervene till an interval after the onset of symptoms, may be due to external disease of the ear, acute or chronic.

Tinnitus or noises in the head comprise as Dr. Dundas Grant aptly remarks "as many varieties of sensation as the patients' powers of description can make them." Tinnitus may be due to impacted wax or disease of the ear, when there is usually associated impairment of hearing and local signs. A *humming* tinnitus worse on lying down and taking food and stimulant is due to venous congestion : relieved by lying down and taking food, is due to anaemia. Tinnitus taking the form of voices or music is auditory illusion due to mental disturbance, and often originates in chronic disease of the organ of hearing. A *pulsating* tinnitus (1) checked by compression of the carotid artery is due to arterial congestion of the middle or external ear ; (2) checked by compression of the vertebral arteries in the sub-occipital triangle—arterial congestion of the internal ear ; (3) audible on auscultating head or ear—probably intracranial aneurysm.

Vertigo and its causes have been dealt with in § 510. Ménière's disease is described in § 524a. Pseudo-Ménière's disease may occur with middle ear catarrh.

Orientation, or as it is sometimes called equilibration, is the function of that branch of the 8th nerve which goes to the semi-circular canals (compare § 502). Disorders of this branch alone are rare, and they cannot well be tested clinically. Disease of this branch or of the semi-circular canals give rise to a sensation described by the patient as giddiness. Horizontal or vertical rotatory vertigo, depending upon which of the ampullæ or semi-circular canals is involved, occurs in association with this condition.

The *prognosis* and *treatment* of these various symptoms depend mainly on the cause in operation. To deal with them individually would be beyond the scope of this work. Nerve deafness is not very hopeful. Any toxic or other cause in operation should if possible be removed, and weak galvanism may be tried. Obstructive deafness is in a large proportion of cases due to middle ear catarrh, which is more hopeful than nerve deafness. A certain amount of good may be done in chronic catarrh by regular inflation, which the patient can be taught to do himself, and regular inhalation of various remedies such as ammonium chloride. Acute middle ear disease requires prompt measures. Hot fomentations, leeches, and incision of the tympanum may be necessary. Syringing with boracic lotion should then be ordered. If the case tend to become chronic employ constant syringing with boracic lotion, varied with astringent solutions and powders, and inflation by the Eustachian catheter. Mastoiditis and other intracranial symptoms demand surgical interference.

§ **615.** The **glossopharyngeal nerve** (the ninth) is the sensory and motor nerve of the pharynx. It supplies the middle constrictor muscle of the pharynx (and the stylo-pharyngeus), and common sensation to the pharynx and the back of the tongue. In the terminal part of its course it contains the taste fibres from the posterior third of the tongue which ultimately go to join the fifth (§ 609).

Anatomy. The 9th, 10th, and 11th nerves arise from a longitudinal collection of cells in the medulla and floor of the 4th ventricle situated beneath or ventrally to the calamus scriptorius. The 9th arises from the upper end, the 10th from the middle and outer part, the 11th from the lower end. The 9th is joined by ascending branches from the lateral column as low down as the 4th cervical segment, and the spinal part of the 11th by branches as low as the 6th cervical segment. The spinal accessory is the only one which supplies voluntary muscles, and it should be remembered that it is the accessory portion of this nerve which supplies the larynx and palate through the vagus.

Paralysis of the glossopharyngeal nerve is practically never met with alone and the exact limitation of its functions has never been defined. Paralysis would be indicated by—(1) loss of sensation (*i.e.*, insensibility to tickling) of the upper part of the pharynx, and (2) some disturbance of deglutition. (3) The sense of taste in the posterior part of the tongue should be tested in suspected cases.

§ **616.** The **vagus** or **pneumogastric nerve** (the tenth) has widespread connections with the respiratory passages, heart, œsophagus, and (through the sympathetic) with all the abdominal viscera. It is also the motor nerve to the larynx, pharynx and palate (by fibres derived from the accessory portion of the spinal accessory, 11th). The involvement of the vagus proper or its nucleus (fortunately not very common) is evidenced by disturbances of the cardiac rhythm, slowing of the respiration, and perhaps by vomiting, hiccough, sighing and yawning. The motor effects are evidenced in the palate, larynx and pharynx.

LARYNGEAL paralysis has already been referred to in § 129. The crico-thyroid is supplied by the superior laryngeal and all the other muscles by the inferior laryngeal branch of the vagus, both of which are really derived from the accessory portion of the spinal accessory nerve. Unilateral paralysis of the larynx need not produce any symptoms, and is nearly always due to some local lesion of a *laryngeal branch* of the vagus. Lesions above the level of the nuclei, *i.e.*, when situated in the cortex of *both hemispheres* or in the descending motor tracts of both sides, always produce bilateral paralysis (as in the case of the eyes), and this bilateral paralysis may be complete or affect only the abductors (a dangerous condition which impedes inspiration). A lesion in the cortex or motor tract of *one side* is unattended by laryngeal paralysis since the opposite cortex can innervate both cords (Horsley and Semon).

§ **617.** The **spinal accessory nerve** (the eleventh) at its origin consists of two portions. (a) The accessory portion rising from the medulla joins the vagus and gives to it its motor functions for the larynx, palate, and pharynx (*vide* § 616). (b) The spinal portion arises from the anterior horns of the first four segments of the cervical cord, and supplies the sterno-mastoid and trapezius (which are also supplied by twigs from the cervical plexus). When the sterno-mastoid is paralysed the patient is unable to turn his head to the opposite side. When the trapezius is paralysed he is unable to shrug his shoulders. This portion of the nerve is often involved in obscure irritation producing torticollis.

The SOFT PALATE is now believed to be supplied from the accessory portion of the 11th through the vagus and the pharyngeal plexus (Fig. 171). Paralysis of the palate is evidenced by (i.) insufficient elevation of one (or both) sides of the palate during phonation when the patient says "ah." Deviation of the uvula to one side is not a proof of paralysis. (ii.) Inability to pronounce words containing g and b. (iii.) Regurgitation of fluids through the nose in advanced cases. As regards the *causes* of paralysis of the palate, (1) it is more often met with in diphtheritic peripheral neuritis than in lesions of the vagus or spinal accessory. (2) A lesion of the nucleus of the accessory portion of the spinal accessory nerve is met with in bulbar paralysis, and occasionally in amyotrophic lateral sclerosis and progressive muscular atrophy. It is also met with occasionally in other spinal lesions, such as tabes. (3) Lesions of the motor tract above the nucleus, a condition that may occur in hemiplegia due to a cerebral lesion.

§ **618.** The **hypoglossal nerve** (the twelfth) is purely a motor nerve to the tongue. Probably the orbicularis oris is also innervated from the *nucleus* of this nerve through the facial. One side may be paralysed in both supra- and infra-nuclear lesions, and is evidenced by the deviation of the tongue when it is protruded *towards* the paralysed side owing to the unbalanced action of the tongue muscles on the healthy side. When the lesion is bilateral the tongue is motionless. (1) Cortical and sub-cortical lesions are very often attended by unilateral non-atrophic paralysis and by hemiplegia. Articulation is only temporarily affected. Lesions producing this condition are mentioned under hemiplegia. (2) Nuclear lesions give rise to atrophic paralysis of one or (more commonly) both sides of the tongue, and the mucous membrane is thrown into folds. In bilateral paralysis speech, mastication and deglutition are much impeded; it is generally part of bulbar paralysis (*infra*). Both nuclei are generally affected together by a degenerative lesion; sometimes one is affected in tabes. (3) In unilateral paralysis of the hypoglossal the lesion is usually situated in the nerve trunk. Sometimes there is a triad of symptoms as first described by Dr. Hughlings Jackson—unilateral hemiatrophy of the tongue, with paralysis of the palate and the larynx on the same side. Such cases are mostly due to a localised pachymeningitis of syphilitic origin involving the trunks of both the hypoglossal and the accessory portion of the eleventh. A fourth symptom is sometimes added—paralysis of the sterno-mastoid and trapezius from involvement of the spinal portion of the 11th. Unilateral lesions of the hypoglossal nerve trunk alone are also met with in parotid and tumours of the neck, and occasionally neuritis.

§ 619. **Bulbar paralysis** (or glosso-labio-laryngeal palsy) is a bilateral, progressive, atrophic paralysis of the lingual, labial, laryngeal, and palatal muscles, due to slow degenerative changes affecting the nuclei of the 12th, and the accessory portion of the 11th nerves. Its description comes suitably at the end of the cranial nerves where several paralyses clinically and anatomically associated have been referred to.

The first *symptom* usually noticed is alteration of the speech, which becomes thick and indistinct, words being run into one another as though the tongue were too large for the mouth. In reality, however, the tongue becomes wasted, and, as in other atrophic paralyses, is the seat of fibrillary tremors. At first the lingual letters only are difficult, but by-and-bye as the soft palate becomes paralysed, words containing gutturals such as "ground" and "grub" are impossible to the patient. Gradually as the tongue wastes the mucous membrane is thrown into folds, and the patient is unable to protrude the organ. (2) The orbicularis oris is simultaneously involved. The patient cannot whistle, and by the falling of the angles of the mouth the expression becomes mournful. The other muscles of the face are unaffected, but he cannot inflate the cheeks owing to the paralysis of the soft palate. (3) The vocal cords are also paralysed, and phonation, difficult at first, becomes by-and-bye impossible beyond a meaningless grunt. (4) Owing to the affection of the soft palate and pharynx, swallowing becomes difficult, and fluids regurgitate through the nose, and the saliva, which the patient cannot swallow, dribbles out of the mouth over the edges of the protruding lower lip. Occasionally the disease is associated with descending sclerosis.

The *diagnosis* in typical cases is not difficult. Certain combined lesions, such as that referred to under the hypoglossal nerve may present a difficulty, but bulbar paralysis is always bilateral. *Myasthenia gravis* (§ 569) resembles it but presents certain generalised symptoms. *Pseudo-bulbar paralysis* is due to bilateral disease of the cerebral cortex in the lower part of the ascending frontal convolutions or about the angle of the internal capsules, but this is much slower and often improves, and is not associated with atrophy of the paralysed muscles.

The *prognosis* is extremely bad. The progress is slow but sure, and a fatal issue invariably results, usually after a course of about one or two years, from complications or the extension of the disease to the vagus or other cranial nuclei.

The *etiology* is obscure. The disease is practically confined to persons at or beyond middle life. It may occur in association with or independently of progressive muscular atrophy, and the morbid anatomy is probably identical with it. The occasional occurrence of an *acute* form of *bulbar paralysis* (due to acute softening analogous to acute anterior poliomyelitis) makes their identity more complete.

Treatment is unavailing, though antisyphilitic remedies may be tried in cases having a history of syphilis.

THE SKULL AND SYMPTOMS REFERABLE TO IT.

§ 620. The **skull** as the brain-case is related to neurology. Scars, exostoses, and the traces of syphilis may be found. The cranial deformities of hereditary syphilis and of rickets are given in the table in § 446 and on p. 27.

The *circumference* of the skull is greatest at the level of the external occipital protuberance and glabella :—

Average in male adult	56 cms.
,, ,, female adult	54 cms.
,, ,, children aged 12 years . .	50 cms.
,, ,, ,, ,, 12 months .	45 cms.
,, ,, infants new born . . .	35 to 40 cms.

The average measurement of the naso-occipital arc in the male adult is 35 cms.

The microcephalic and macrocephalic skulls, facial asymmetry, narrow forehead, scapho-cephalic skull (with high vertex and narrow transverse diameter), protruding teeth, or a high narrow arch of the hard palate are all regarded as degenerate or neuropathic evidences; but they are not infallible signs.

External landmarks of the brain. The chief cerebral structures to locate are the fissure of Rolando, and the fissure of Sylvius. The *fissure of Rolando* is a most important landmark, and may be found by drawing a line from the root of the nose to the occipital protuberance. Then mark a point half to three-quarters of an inch behind the middle of this line, and draw a second line from that point downwards and forwards at an angle of 67 degrees; the second line will lie over the fissure. This is conveniently done in practice with soft metal joined at the required angle. The fissure of Sylvius is found by drawing a line from the external angular process of the frontal bone to the occipital protuberance. The fissure starts at a point $1\frac{1}{8}$ inch behind the external angular process. The horizontal ramus of the fissure lies beneath a line drawn from this point to the parietal eminence.

Sir Wm. Macewen attaches importance to the percussion of the skull as an aid to the diagnosis of intracranial tumours.

§ 621. **Hydrocephalus** is a distension of the ventricles of the brain with fluid. It occurs in two forms, (a) congenital or infantile, and (b) acquired or secondary hydrocephalus.

(a) Typical cases of CONGENITAL HYDROCEPHALUS may be recognised by the shape of the head which is enlarged and dome-shaped, projecting all round, and beyond the bones of the face. The fontanelles are very wide, and Wormian bones may fill up the gaps between the cranial bones. The face in proportion to the size of the head seems to be extremely small. The orbital plates of the frontal bones are pushed downwards, so that there is exophthalmos and the eyelids do not cover the sclerotics. The condition dates from birth; it may exist before birth sufficiently to obstruct labour, or it may be so slight as not to be noticed until the child is 1 or 2 years old. The circumference of the head of a child of four may reach as much as 25 or 30 inches. The general symptoms consist of bodily weakness and developmental delay, and backward mental condition, which shows itself in the slighter cases as a constant peevishness, and in the graver cases as idiocy. In some cases the intellect is quite normal. Spina bifida, talipes, encephalocele and other developmental deformities may accompany hydrocephalus. The *causes* of this form of hydrocephalus are not known.

In slight cases the *diagnosis* may have to be made from rickets, in which there are (1) a bossed and square shape of the head, (2) a flattened instead of a dome-shaped vertex, (3) evidences of rickets in the other bones of the body.

Prognosis. Most cases of congenital hydrocephalus die within the first five years of life. Moderate cases sometimes live until the 12th year. Mild cases of hydrocephalus sometimes do not deteriorate further: the patient lives without other symptoms perhaps for the normal span of life.

(b) The symptoms of ACQUIRED HYDROCEPHALUS are less decided: they come on insidiously at any age and are associated with those of the causal condition. The head does not enlarge if the disease supervenes after closure of the fontanelles, but there are symptoms of cerebral compression (§§ 540 and 544), and sometimes gradual blindness due to optic neuritis.

Causes. Acquired or secondary hydrocephalus mostly depends on some obstruction of the veins of Galen, such as that produced by a tumour at the base of the brain, or in the 3rd ventricle, or the iter. It may also result from intracranial inflammation, especially post-basic meningitis (*q.v.*). Pressure on the middle cerebral or straight sinus, into which the venæ Galeni empty, has the same effect (§ 544).

The *treatment* of congenital hydrocephalus is not hopeful. Drugs are powerless to promote the absorption of the fluid. In the milder cases the treatment resolves itself broadly into a general strengthening or tonic treatment. In other cases "lumbar puncture," similar to that successfully used in tuberculous meningitis, has been employed with a measure of success in hydrocephalus. A smooth fine aspirating needle is introduced between the 3rd and 4th lumbar vertebræ, a little to one side of the middle line, to the depth of 2·5 centimetres in children, and 5 centimetres in adults. One to 1½ oz. has been removed at a time (Quincke). It is especially worthy of trial if pressure symptoms are present. The treatment of acquired hydrocephalus is not much more promising unless the cause is removable.

CHAPTER XX.

EXAMINATION OF PATHOLOGICAL FLUIDS AND CLINICAL BACTERIOLOGY.

IN this chapter the methods of obtaining various pathological fluids, how to examine them, and their characters will be briefly described, and in conclusion, an epitome of the chief bacteriological data required for clinical work will be given.

§ 622. **Methods of examination and characters of pathological fluids.**—METHODS OF OBTAINING VARIOUS PATHOLOGICAL FLUIDS. Fluids are obtained by puncture with an ordinary hypodermic needle or with exploring needles, which are made specially strong and with somewhat larger calibre to permit of turbid fluid entering them. The needle must be boiled before use and the skin thoroughly cleansed with soap and water, ether, and 1 in 30 carbolic acid. Cocaine or eucaine may be painted over the spot or injected in the vicinity, or the part may be frozen with the ethylchloride spray.

The *pleural* cavity is best explored at the ninth space just behind the posterior axillary line, or at the site where localised dulness is present. Paracentesis thoracis is described on p. 182.

Pericardial fluid is obtained by inserting the needle ½ to 1 inch to the left of the sternal margin, in the 4th or 5th interspace. Unless we are certain that the pericardium is very full of fluid, it should never be attempted. Paracentesis pericardii has been described on p. 69.

The *peritoneal* cavity may be explored for fluid in the middle line, to avoid blood vessels, or at the side, above the anterior superior iliac spine. The puncture must be made over a dull area. **Paracentesis abdominis** for the removal of ascitic fluid is performed with an ordinary trochar and canula. A many tailed bandage is first placed under the patient, and the tails are tightened up one by one to maintain the abdominal pressure as the fluid drains away. Without this precaution the patient may suffer collapse from the rapid dilation and congestion of the splanchnic area. Some advise a small preliminary incision through the skin; or the trochar may be thrust through the skin in the middle line. The fluid drains away through a long rubber tube into a pail by the side of the bed on the floor. Before puncturing in the middle line care must be taken to ensure the bladder being empty. Southey's tubes may be employed instead of the trochar and canula; they act as efficaciously, though more slowly.

Lumbar puncture is employed chiefly for diagnosis of the character of the fluid in increased exudation into the sub-dural space, and also for relief of certain forms of hydrocephalus. Children should be anæsthetised; for adults local anæsthesia alone is sufficient. Employ an antitoxin needle without any syringe. The patient lies on the right side with the knees drawn up and the back bent towards the operator. Find the 3rd lumbar vertebra, and with the thumb of the left hand to guide, push the needle deeply into the space between the 3rd and 4th lumbar vertebræ, just to the right of the mid-line, in an upward and inward direction to a depth of one-third to one inch according to the age and musculature of the patient. The fluid runs out drop by drop and is caught in a sterilised test-tube.

How to examine Pathological Fluids. Fluid which has been withdrawn from the pleural, pericardial or peritoneal cavity, or from a cyst, should be placed in a conical vessel and allowed to settle, and its physical and chemical characters noted (*see* table, p. 1117). The *colour* should be observed, whether clear, turbid, or red, as with blood, or opaque from abundant cellular constituents, or excess of fat as in chylous exudations. The *reaction* is usually alkaline. The *odour* tells little as a rule except in the case of cysts which have been in the vicinity of the intestinal canal. The *consistence* is usually watery: if viscid, mucin is present, as in a distended gall-bladder or an ovarian cyst: clotting after standing indicates the presence of fibrin. The *specific gravity* must be noted. The *deposit* must be examined under the microscope. Blood cells, white and red, may be found; the former being more readily seen by the addition of acetic acid, which brings out the nuclei. Cancer cells may be found in malignant disease of the wall of the cavity, and are distinguished from epithelial cells by being large and occurring in groups. The discovery of hooklets (Fig. 75, p. 457) or pieces of lining membrane of hydatid cysts, crystals of cholesterin, leucin or tyrosin, or various germs, or the amœba of dysentery, or actinomyces, may clear up a difficult diagnosis. Fatty acid crystals in groups favour the diagnosis of cancer.[1]

The *chemical examination* is then proceeded with after filtering the fluid. Albumin, nucleo-albumin and mucin are tested for as in urinary examination. For quantitative examination of albumin the fluid is first diluted to a specific gravity of 1008 and acidified with acetic acid, then measured in Esbach's tube (§ 279). To detect urea first remove any albumin by heating and filtering; then evaporate the fluid to small bulk and test as in urine. To detect sugar render the fluid slightly acid, boil, filter, evaporate to small bulk and test as in urine.

When the fluid is suspected to come from a pancreatic cyst, it is tested to ascertain if it can digest albumin in an alkaline medium. The suspected fluid is added to milk; after the casein is precipitated the Biuret test is tried. (A few drops of dilute solution of copper sulphate, and excess of caustic potash, causes a rose red hue in the presence of peptones.) If present it proves that the fluid has a peptonising power, and nothing except pancreatic fluid can peptonise in an alkaline medium. In cysts of some standing the power of peptonising may be lost owing to the destruction of the trypsin.

Characters of the Pathological Fluids (table below). In the pleural, pericardial, and peritoneal cavities, inflammatory effusions (exudates) are difficult to distinguish from dropsical effusions (transudates). Cardiac dropsy is more albuminous than renal dropsy. A fluid containing more than 4 per cent. of albumin is probably of inflammatory origin: a fluid with

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[1] Simon, "Clinical Diagnosis," p. 466, Kimpton, London, 1896.

TABLE OF PHYSICAL AND CHEMICAL CHARACTERS OF PATHOLOGICAL FLUIDS.

Constituents, etc.	Serous exudation as in ascites or pleurisy.	Transudation.	Hydatid cyst.	Pancreatic cyst.	Ovarian cyst.	Hydronephrosis.	Cerebro-spinal fluid (normal).	Distended gall-bladder.
Colour.	Turbid yellow.	Clear yellow or greenish yellow.	Clear and colourless or opalescent.	Colourless. Often mixed with blood.	Variable—clear yellow to black.	Clear and watery.	Clear.	Clear or stained with bile.
Specific gravity.	Usually over 1018.	Rarely over 1015.	1006—10.	Variable—often 1068—10.	From 1002—1055 (low sp. gr. points to cyst of broad ligament).	1008—1020.	1006—7.	Low.
Spontaneous coagulability.	Very marked. Clots in 24 hours or earlier.	Slight. May clot after long standing.	Absent.	Absent.	Absent. Consistence variable — watery to colloid.	Absent.	Absent.	Absent.
Chemical composition.	Alkaline. Much albumin, 40% (serum alb. and globulin). Small quantities { Sugar. Uric acid.	Alkaline. Albumin 5—25% (serum alb. and globulin).	Alkaline. No albumin or negligible quantity. Much sodium chloride. Succinic acid.	Digests albumin in alkaline medium. Serum albumin present	Alkaline. Albumin (serum alb. and globulin, and *metalbumin*).[1]	Traces of albumin, urea and uric acid.	½ to 1 albumin. Small amount pyrocatechin, a reducing agent.	Mucin present may have bile.
Microscopic examination.	Very little debris. { red blood cells. leucocytes. endothelial cells.	Even less debris than in serous exudation similar constituents.	Characteristic hooklets, scolices, cyst membrane, hæmatoidin crystals.	Often cholesterin crystals.	Cylindrical ciliated epithelial cells, squamous cells, colloid concretions, red blood cells, leucocytes, fat globules, cholesterin, fatty acid crystals, and hæmatoidin.	Sometimes renal epithelial cells, uric acid crystals.		
Remarks.	May be variable quantity of pus present. Occasionally it is red with blood as in cancer.[2]	May be sanguineous, and, rarely, chylous.	Diagnosis made by microscopic examination.	The ability to digest albumin is a positive test; but in very old cysts the power is lost.	Metalbumin and ciliated cells the diagnostic points.	Only diagnostic point is presence of renal cells, and these may be absent.	In meningitis the fluid is cloudy, with more albumin. Micro-organisms found aid diagnosis.	

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[1] Metalbumin is thus tested for. Acidify fluid with acetic acid, boil and filter to remove other forms of albumin. Add to the filtrate 3 times its bulk of alcohol. Stand for 24 hours. Filter. Squeeze out precipitate and suspend in water. Filter. Filtrate has following reactions. (i.) On boiling it becomes turbid, but no precipitate falls; (ii.) on adding acetic acid no precipitate forms; (iii.) on adding acetic acid and strong sulphuric acid a violet colour is produced; (iv.) on adding acetic acid and pot. ferrocyanide the fluid is thick and yellow.

[2] Ascitic fluid usually shows the characters above described, "hæmorrhagic ascites," where blood is found in the fluid, occurs chiefly in cancerous peritonitis, and has been considered a fatal sign. Middleton reports cases of chronic peritonitis associated with hæmorrhagic ascites in alcoholics, who recovered. *Lancet*, 1899, ii., p. 1286.

less than 2½ per cent. of albumin is probably dropsical. Blood in distinct amount in these cavities is highly suggestive of cancer, but has sometimes been found with tuberculous disease of the pleura and with peritonitis associated with cirrhosis of the liver. In such a case, examination of the deposit, revealing cancer cells or tubercle bacilli, would settle the diagnosis. A few blood cells, sufficient to give the fluid a rosy tinge, may occur with simple acute inflammation. The character of the cells in a pleuritic fluid may aid the diagnosis of the cause of the effusion (cyto-diagnosis); thus, an excess of lymphocytes points to tuberculous pleurisy: the predominance of polynuclear cells points to septic or other causes. The special characters of the various fluids and cysts are given in tabular form; but a few words should here be said about the cerebro-spinal fluid.

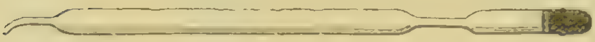

Fig. 175.—PASTEUR PIPETTE.

CEREBRO-SPINAL FLUID should be clear, colourless, containing ½ per cent. albumin, and Fehling's solution is reduced (? by pyrocatechin). When the fluid which runs out is turbid it indicates meningitis of some kind, and cover-glass films should be made to study the organisms present. There may be found the pneumococcus, or the diplococcus intracellularis of cerebro-spinal meningitis, or the diplococcus of infantile post-basic meningitis. In negative cases where tubercle is suspected, a guinea-pig may be inoculated with the fluid. Cyto-diagnosis or diagnosis by the cellular elements present in the fluid is often very useful in cerebro-spinal fluid. In tuberculous meningitis lymphocytes predominate, in pneumococcic and cerebro-spinal meningitis, the polynuclear leucocytes are in excess. In tabes dorsalis and some chronic syphilitic meningeal affections the lymphocytes also predominate.

§ 623. **Clinical Bacteriology.** The subject of serum-therapy and immunity has been dealt with in Chapter XV. Here will be described in brief outline the methods of detecting and the appearance of the various microbes commonly met with in clinical work. For laboratory work the student must consult a special treatise.

The necessary APPARATUS consists of (1) slides and cover-glasses thoroughly cleansed as described in the examination of blood (§ 396). (2) A pair of Cornet's forceps made so that they hold the cover-glass *horizontally* when laid on a table. (3) Two platinum points fixed to the end of glass rods. (4) Sterile swabs (which are said to be obtainable from any medical officer of health). To make them, twist a piece of cotton wool round the end of a stiff copper wire about eight inches long. Place this in a test tube and plug its mouth with cotton wool; the end of the wire should pass through the plug. The tube, with its contained swab, should then be sterilised by heat. (5) Stains—methylene blue, eosin, carbol-fuchsin, Gram's stain and other reagents. (6) A good microscope, including a $\frac{1}{12}$th inch oil-immersion lens. (7) In some cases the Pasteur pipette is of use (Fig. 175; and see blood, § 400). It is a straight glass tube drawn out to a point at one end, with a piece of sterilised cotton wool inserted at the other end, just below which is a constricted or narrow portion. It can be used to remove drops of fluid, by sucking up through the cotton wool, or for conveying away a drop of blood serum for the Widal reaction or other test. In the latter case the point is sealed up in a flame, and the constricted portion is likewise sealed and broken off beyond. (8) A few tubes of nutrient media, such as gelatine, agar, and blood serum, and a small incubating oven are useful in many instances.

METHOD OF EXAMINATION SUITABLE FOR CLINICAL WORK. (1) With a looped platinum wire which has been thoroughly cleansed by heating in the Bunsen burner or spirit flame, select a minute portion of the material to be examined and smear this on the cover-glass with the end of the wire, or place another cover-glass on the top of the first and slide them apart (see blood, § 399). A thin film is thus formed. Take care not to touch the cover-glasses with the fingers. If the material be too thick dilute it with a drop of sterilised water. (2) Let the film dry in the air, or by holding it high above the flame, and fix by passing it rapidly through the flame three times. (3) Put a drop or two of the selected stain (previously filtered) on the cover-glass, or float the cover-glass, film downwards, in a watch-glass containing the stain. The time of leaving the film in the stain depends on the stain employed, but in most cases 1 to 3 minutes is sufficient. (4) Rinse the cover-glass *thoroughly* in water and dry by blotting paper or gentle heat high over the flame. After using the carbol-fuchsin stain the cover-glass should be passed through a 1 in 4 solution of sulphuric acid (which decolorises everything except the tubercle bacillus and certain other microbes), then rinsed again in water and dried. The preparation may then be mounted in a drop of water and examined, or mounted in Canada balsam as a permanent preparation. A drop of cedar oil is placed on the cover-glass and the oil-immersion lens employed.

Motile Organisms may be examined unstained and their movement readily seen. When examining unstained preparations remember to reflect the light with the plane mirror of the microscope placed in a slanting position, without the Abbé condenser. Place a drop of sterilised water on a cover-glass and touch it with the platinum wire, which has previously been dipped in the material to be examined. The glass may be placed over the hollow of a hollow slide, thus forming the "hanging-drop"; but the movements can be as readily seen in the hollow or cell formed by drawing a ring of vaseline round the edge of the cover-glass.

Gram's Method.—Certain organisms possess the characteristic reaction that their stain is fixed by iodine. Place the cover-glass, film side downwards, in a solution of aniline-gentian-violet for 5 to 8 minutes. This is prepared by adding one part of aniline oil to twenty of distilled water, shaking thoroughly and filtering the mixture; to this add drop by drop a concentrated and filtered alcoholic solution of gentian violet, in the proportion of one part of aniline water to nine parts of the stain. (2) Place the film in a solution made up of one gramme of iodine and two grammes of potassium iodide in 300 c.c. of water. This acts as a mordant and fixes the stain in the microbe. The film becomes black. (3) Place the film in alcohol till it becomes a faint grey, everything except the microbe becoming decolorised. (4) Wash, dry, and mount as usual; or counterstain the other structures with eosin before mounting.

a. The **sputum** is examined for tubercle bacilli, the pneumococcus, the influenza bacillus, oïdium albicans of thrush, the bacillus of plague in cases of pneumonic plague, anthrax, the actinomycosis fungus, and the micrococcus tetragenus (see below). Pyogenic organisms are found when pus is present.

(1) The **tubercle bacillus** is slightly curved, measuring 2 to 5 μ long; three go to the diameter of a red blood corpuscle. Often two lie together at an angle, like the letter V. Clear spaces resembling spores are often seen within them after staining. The tubercle bacillus is present in the *sputum* in all cases of active pulmonary tuberculosis. It is usually most plentiful in the thicker and most purulent part of the sputum. If hæmoptysis be present, select a dark red clot after the attack has subsided. Take the minutest portion possible and press it between two perfectly clean cover-glasses. Separate these by sliding the one over the other, and fix the films thus obtained by passing them two or three times through the flame of a spirit lamp. When the bacillus is hard to find (as in cases where very few are present) boil about 100 c.c. of the sputum with double the amount of water, to which 6 drops of a 10 per cent. solution of caustic potash have been added. Add water from time to time to allow for evaporation; then set aside for 24 or 48 hours, or centrifugalise, and examine the sediment for the tubercle bacillus.

The tubercle bacillus may be detected by its characteristic staining reaction. In the Ziehl-Nielsen method, which is the best, carbol-fuchsin is dropped on the cover-glass film, which is then heated over a flame until the liquid steams. Or heat the stain in a watch-glass and drop the cover-glass, film downwards, into it, and leave in for 3 minutes. Then decolorise in 33 per cent. or 25 per cent. sulphuric acid, and wash the glass in water. If a counter-stain be desired, in order to examine other elements of the sputum, leave the glass in a watery solution of methylene blue for 2 or 3 minutes. Then wash in water, dry, mount, and examine.

Other microbes which stain by the Ziehl-Nielsen method are the leprosy and the smegma bacillus. Leprosy bacilli occur in clumps, never isolated like tubercle. The smegma bacillus may readily be mistaken for tubercle in the examination of the urine. To distinguish it, place the cover-glass in absolute alcohol for 3 hours, then ¼ hour in chromic acid. Stain as usual, and wash in alcohol after decolorising in acid. The smegma bacillus is thus decolorised.

(2) **The pneumococcus** (Fraenkel's pneumococcus) is a coccus usually of lanceolate shape, often in pairs (diplococcus), sometimes in short chains. A capsule surrounds it which is less deeply stained in stained preparations and is absent in cultures. Sometimes more than two cocci occur within one capsule. They cause croupous pneumonia, but are also found in healthy saliva. Select a rust-coloured part of the sputum when the disease is at its height. It is found in the lungs in the red hepatisation stage of the disease. The pneumococcus is easily *stained* by ordinary aniline dyes. It can also be seen by Gram's method, which distinguishes it from Friedlander's pneumo-bacillus (also found in pneumonic sputum), the latter being decolorised by Gram. By the carbol-fuchsin method the cocci should be deeply stained and the capsule a fainter red, or altogether unstained.

Plate III

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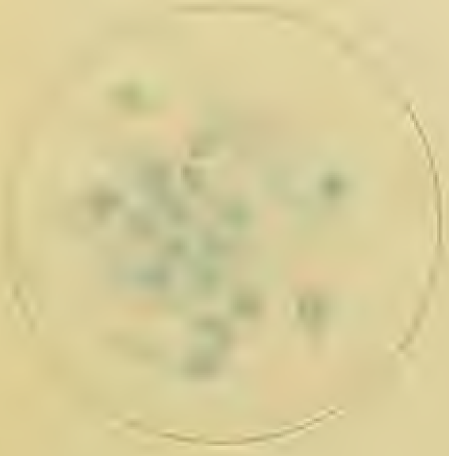

FIG. 1.—*Tubercle bacilli in sputum* × 800.
Stained by carbol-fuchsin and methyl blue.

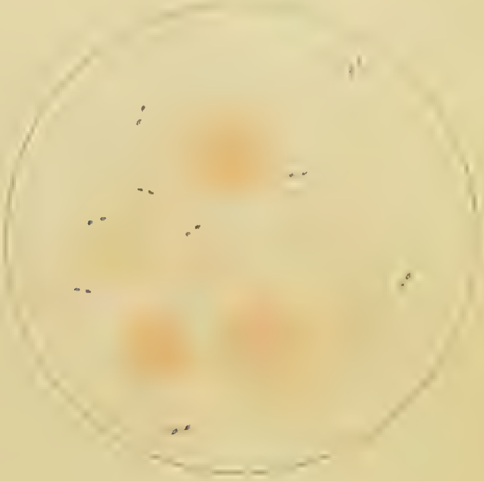

FIG. 2.—*Pneumococcus in sputum* × 800.
Stained by Gram's method.

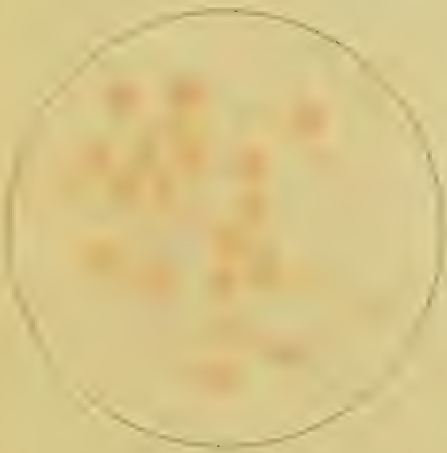

FIG. 3.—*Streptococcus in pus* × 800.
Stained by Gram's method.

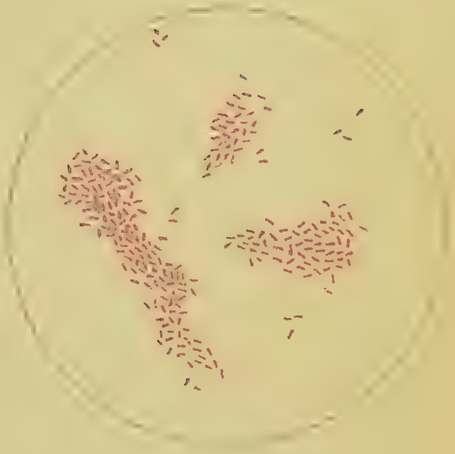

FIG. 4.—*Typhoid bacilli* × 800.
Stained by carbol-fuchsin.

(3) The **influenza bacillus** (Pfeiffer's bacillus) is found in the sputum and nasal mucus of patients suffering from influenza. It occurs as a minute rod, not quite 1 μ long, lying in pairs or groups, and may be mistaken for diplococci. It stains with ordinary dyes. It may also be found in the blood at the acme of the disease.

(4) The **micrococcus tetragenus** occurs as cocci in groups of four in a capsule. It is found in the sputum coming from phthisical or bronchiectatic cavities. It stains with the ordinary dyes and with Gram's method.

(5) The presence of the **actinomycosis** fungus is suspected when small sulphur coloured granules, visible to the naked eye, are seen in the sputum or in purulent discharge from tumours. Such granules under the microscope are seen to consist of a large number of threads radiating out from a centre like a fan, each thread having club-shaped ends. They stain well with Gram's method. The pus has a characteristic greenish yellow colour.

b. A **false membrane** occurring on the throat or palate must be examined for the presence of the **diphtheria bacillus.** Taking care not to touch the lips, cheek, or mouth inside, touch the suspected patch with a sterilised swab or brush or remove a piece of the membrane with a platinum loop or forceps. Transfer this to a sterilised test tube, or spread thinly on a cover-glass, fix, stain and examine. Frequently so many micro-organisms are present that it is impossible to make out satisfactorily the diphtheria bacillus. In such cases cultures must be made by inoculating tubes containing blood serum or coagulated egg albumen with the platinum needle or swab. On such media, kept at body temperature, the diphtheria bacillus grows rapidly in characteristic colonies of punctiform spots which fuse into opaque, uneven streaks. The growths can be examined under the microscope within 24 hours.

The bacillus is known as the Klebs-Lœffler bacillus; it is of variable size and shape, average 2 μ long. It is straight or slightly curved; some of the rods are knobbed, thicker at one end. Some stain uniformly, others irregularly. They lie in clusters, never in chains; there are no spores. Streptococci are frequently found with it, but are no index of the severity of the case. Lœffler's methylene blue is recommended as the most suitable stain, because by its use the characteristic beaded appearance or polar staining of the rods is well shown, but any other basic stain may be used. It also stains by Gram's method.

Hoffman's *pseudo-diphtheria bacillus* resembles the true diphtheria bacillus, but it stains more deeply, and has not the polar staining which gives to true diphtheria its characteristic club shape. In difficult cases the distinction can be drawn only after cultures are made on alkaline potato—the pseudo-bacillus grows cream coloured colonies in two days, the growth of the Klebs-Lœffler is almost invisible.

c. **Pleural effusion** may be examined after fixing and staining on a cover-glass or a slide, the latter giving a larger area for examination. Serous effusions show few or no germs: the diplococcus pneumoniæ is almost the only germ which has yet been found in a serous pleurisy. The tubercle bacillus is very rarely found either in serous or cloudy effusions, even when the condition is undoubtedly tuberculous in origin. The rheumatic diplococcus may be present, but as yet cannot be differentiated by clinical methods, only by inoculation of animals. The germs found in purulent effusions are the diplococcus pneumoniæ and the streptococcus and staphylococcus.

d. **Pus** obtained from any part of the body should be examined for the streptococcus, the staphylococcus, and the gonococcus.

(1) **Streptococci** are large cocci occurring in long chains.

(2) **Staphylococci** are large cocci occurring in small groups or bunches. Both are pyogenic organisms and are found in pus in various situations. They stain with ordinary dyes and with Gram's method.

(3) The **gonococcus** appears as a diplococcus, the paired cocci facing each other with concave surfaces. They are usually found inside the pus corpuscles lying in groups. They are most readily found in the pus coming from a gonorrhœal, urethral or conjunctival discharge. They stain with the ordinary dyes, and not with Gram's method. It is well to use a counterstain, such as eosin, which stains the corpuscles in which the gonococci lie.

(4) Buboes may require to be examined for the bacillus of **Plague** (bacillus pestis). The bacillus is short and thick, 2·3 μ long by 1·7 μ broad. It often lies in pairs and so may be mistaken for a diplococcus. In cultures it forms chains and may present remarkable yeast-like forms. It stains with the ordinary dyes; presenting characteristic polar staining with methylene blue, the central part remaining pale. The most valuable tests for the plague bacillus are its pathogenic action on certain animals and its characteristic growth on bouillon—"stalactite" formations growing downwards from the film on the surface of the bouillon. The bacillus of plague occurs not only in buboes, but in the sputum in pneumonic plague, in the spleen and blood, and in the bodies of rats, which are believed to be the chief means of conveying the disease. When a plague bubo is suppurating, no bacilli can, as a rule, be found. The best way then is to puncture the middle of a bubo and draw off a few drops of serum, but if the bubo is already suppurating, in order to get bacilli you must insert the needle into the margin of the swelling. The pus discharged from buboes generally contains no bacilli.

The **malignant pustule** due to anthrax can rarely be shown to contain the anthrax bacillus. In doubtful cases examine the blood (see below).

The purulent discharge from a tumour caused by the actinomyces fungus contains tiny granules, which are described above under the sputum.

e. It is difficult to examine the **stools** for any particular germ because of the enormous number of non-pathogenic germs contained in the fæces. Two, however, may be mentioned here—the cholera vibrio and the amœba of dysentery.

The **cholera vibrio** (Koch's comma bacillus) is obtained from the mucoid masses in the choleraic stools; but in at least half the cases no definite result is gained by simple staining methods, and the organism can be recognised only by submitting it to a number of cultural and other tests. The vibrio is a curved rod, slightly shorter than the tubercle bacillus. They lie in rows, end to end, pointing in one direction. They stain with ordinary dyes, not by Gram's method.

The **amœba of dysentery** is a spherical body, sometimes of a pale-greenish colour, refracting the light strongly. It is 30 to 40 μ diameter;

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has a granular endoplasm and a clear ectoplasm, and is motile. The amœba should be looked for in the stools at once while the discharges are still alkaline. The glass slides, cover-glass, and the microscopic stage should be warmed. A drop of mucus from the stools, or a scraping from the abscess wall is diluted with warm saline solution and placed on the slide. The amœba will then be seen actively moving. The amœba is easily killed ($\frac{1}{5000}$th solution of quinine arrests the movement), and if examined when the stools are acid the amœba will have no movement.

f. The **urine** may require to be examined for the tubercle bacillus, the gonococcus, the typhoid bacillus, and the colon bacillus. The tubercle bacillus is described under sputum, and the gonococcus under pus above. The diplococcus of rheumatism has been found in the urine, but requires experimental testing for its detection.

The tubercle bacillus is described under sputum. It is sometimes very difficult to detect in urine. It is best to draw off the urine by a sterilised catheter to prevent the entrance of the smegma bacillus. Stand the urine for 24 hours in a conical glass, or centrifugalise it. Examine the sediment carefully, staining, etc., as with sputum.

The **typhoid** bacillus (Eberth's bacillus) is short, thick, and mobile, with rounded ends. It is 3 to 4 μ long, and 1 μ broad, and is flagellated. Sometimes several rods lie end to end. It may be very difficult to diagnose from the bacillus coli communis, but is known by its characteristic cultures. It stains with ordinary dyes, and not with Gram's method.

The typhoid bacillus is the cause of enteric fever and occurs in the spleen, in the blood, in the rose-coloured spots, in the urine, and in the stools. However, one is not likely to succeed in finding them by microscopic examination except from culture growths, and expert knowledge is required to succeed in obtaining a pure culture. The ordinary clinical method of proving the presence of enteric fever is the Widal serum test. The patient's finger is carefully sterilised and pricked near the nail with a sterilised needle. and the blood is drawn off by a Pasteur or Wright's pipette (§400 and Fig. 175). This is then at once sealed and taken to the laboratory.

(i.) *Widal's reaction* depends upon the fact that the blood serum of a patient suffering from enteric fever contains antitoxin, which causes typhoid bacilli to run together in clumps, to agglutinate and to become immobile. This reaction is not found until the second week of the disease; it may go on to the third week after the cessation of pyrexia, and it may be retained for months after the disease. For the proper performance of this test it is necessary to have (1) a fresh culture of typhoid bacilli in bouillon, not more than 24, not less than 12 hours old. (2) Mix a loopful of the bacilli with a little sterile water and examine under the microscope to make certain that they are actively motile. (3) Having ascertained that the bacilli are active and discrete, prepare a mixture of bouillon and blood serum in a proportion of 1 in 30 (never less than 1 in 20). Have two watch glasses ready. Into one place 29 drops of the bouillon, measured with the loop of a platinum wire. Then take the pipette in which the blood has been brought from the patient; shake away the clotted blood from the serum, break off the end of the pipette and place the serum in the second watch glass. Next, take out with the platinum wire one loop of serum and add it to the bouillon in the first watch glass, and thoroughly mix. (4) At intervals of ten, twenty, thirty, and sixty minutes examine a drop or two of the mixture under the microscope. In a typhoid case the reaction is usually present within a quarter of an hour. The reaction should be complete within an hour if the serum is that of a typhoid patient. If the reaction is negative try again. Clumping of the bacilli may occur after several hours contact with serum other than that of a typhoid patient. Recent workers have found that this test is not absolutely pathognomonic, but must be regarded as a most valuable confirmatory sign.

(ii.) The slower *sedimentation test* may be employed. Add a few drops of the suspected serum to a cloudy bouillon culture of Eberth's bacillus: and if there is a precipitate at the bottom of the tube after a few hours, and clear bouillon above, the diagnosis of typhoid is established. The principle underlying this test is the fatal action of the serum of an individual suffering, or convalescent from, enteric fever, on the Eberth bacillus. The serum causes the bacillus to become immobile and to agglutinate.

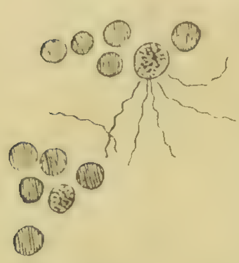
Fig. 176.—Spirillum of Relapsing fever.

For the sedimentation test it is useful to have a control experiment with normal blood serum. An apparatus has been devised by Hutchinson with 2 tubes clamped side by side on a stand. In one of these is shown a sedimentation reaction with the suspected serum: in the other, a cloudy bouillon without a precipitate after the addition of normal serum.

(iii.) On account of the difficulty of obtaining fresh typhoid cultures many physicians cannot make use of the Widal reaction test. Widal, however, pointed out that the agglomeration test can be equally well obtained when the typhoid serum is in contact with dead typhoid bacilli killed by exposure to a temperature of 60° C. Professor A. E. Wright[1] described how the practitioner abroad can carry about with him without risk dead cultures of both typhoid and Malta fever germs, and thus readily diagnose the presence or absence of the respective fevers.

(iv.) The *Wyatt-Johnson method* is also useful for physicians who live out of reach of laboratories. A drop or two of the patient's blood is sent on a piece of non-absorbent paper to those who can examine it. The blood is dissolved in distilled water, and the Widal test can be tried.

The **colon bacillus** (b. coli communis) may also be found in the urine, causing cystitis, pyelitis and pyelonephritis, apart from any other pathogenic germ. It has also caused suppurative peritonitis. It closely resembles the typhoid bacillus, but is somewhat shorter. It is motile, but has fewer flagellæ than the typhoid bacillus. It stains with ordinary dyes, not with Gram's method. It can only be distinguished by cultural and other tests.

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[1] *Brit. Med. Journ.*, 1897, vol. i., p. 1214.

g. The **blood** may be examined for certain parasites—malaria, trypanosoma, filaria, kala-azar, etc. (§ 400). The pyogenic organisms, the typhoid bacillus, the rheumatic diplococcus, the anthrax bacillus, and the micrococcus of Malta fever have been found in the blood. The spirillum of relapsing fever is always present in the blood in the febrile periods (Fig. 176). Kala-azar and some other microbes are difficult to detect unless a large quantity of blood is available, and for this purpose the blood may be drawn off with a hypodermic syringe from the spleen or liver.

In suspected cases of **anthrax** examine the blood, as the anthrax bacillus is only rarely obtained from the pustule or local lesion, from which the disease spreads to infect the blood throughout the body. The bacillus is one of the largest, about 5·2 μ long. When culture growths are examined spores are seen inside the filamentous segments and lying around. It stains with the ordinary dyes and with Gram's method.

The **Micrococcus Melitensis** is the cause of Malta fever. It is a minute coccus ($\frac{1}{3}$ μ), motile, sometimes lying in chains. It can be obtained by puncturing the spleen with a thin sterilised hypodermic needle with due precaution, and withdrawing a little blood. This is a dangerous method, and the presence of the disease may be diagnosed by the agglutination tests (see typhoid, test c). The coccus stains with the ordinary dyes and not by Gram's method.

h. The **cerebro-spinal fluid** may contain pyogenic germs, tubercle bacilli, the pneumonia diplococcus, the diplococcus of epidemic cerebrospinal meningitis, and that of posterior basic meningitis, the trypanosoma, and more rarely, the bacilli of typhoid, anthrax, or the actinomycosis fungus. The cerebro-spinal fluid is obtained by lumbar puncture (§ 622), and a film is made and fixed in the usual way. Centrifugalisation and examination of the sediment is advisable when looking for the trypanosoma.

The diplococcus of epidemic cerebro-spinal meningitis and post-basic meningitis is known as the **diplococcus intracellularis** of Weichselbaum. The cocci lie in pairs, within the pus corpuscles, thus resembling the gonococcus. It stains with ordinary dyes and not with Gram's method.

The **diplococcus of posterior basic meningitis**, which occurs in infants, is found in the cerebro-spinal fluid, but is rarely found within the pus cells, and has not the lanceolate shape of the pneumonia diplococcus. It tends to be grouped in pairs, resembling sarcinæ or tetragenus. It stains with ordinary dyes, and not with Gram's method. It has more vitality than the diplococcus of epidemic cerebro-spinal meningitis, living for several weeks on agar cultures.

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